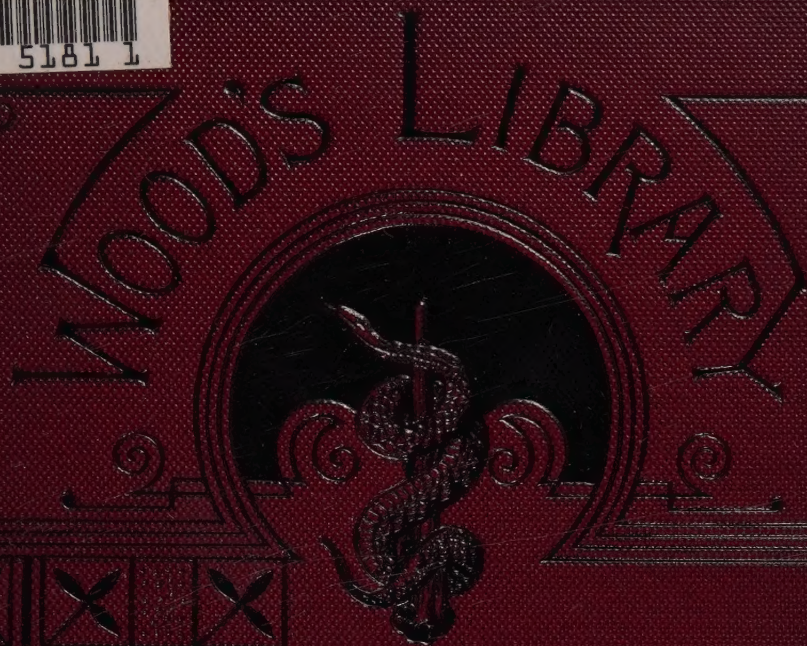


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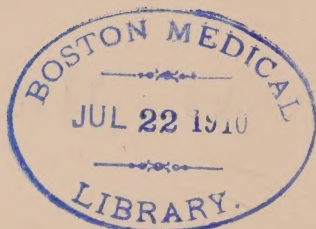
BY
Dr. HERMANN EICHHORST,
PROFESSOR OF SPECIAL PATHOLOGY AND THERAPEUTICS AND DIRECTOR OF THE
UNIVERSITY MEDICAL CLINIC IN ZURICH

VOLUME II
DISEASES OF THE DIGESTIVE, URINARY, AND SEXUAL APPARATUS

ONE HUNDRED AND SIX WOOD ENGRAVINGS

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TABLE OF CONTENTS.

DISEASES OF THE DIGESTIVE APPARATUS.

PART I.

	PAGE
DISEASES OF THE BUCCAL CAVITY AND SALIVARY GLANDS, THE SOFT PALATE, AND THE PHARYNX,	1-26
1. Catarrhal inflammation of the buccal mucous membrane.	
Catarrhal stomatitis,	1-4
2. Ulcerative stomatitis,	4-7
3. Aphthæ. Aphthous stomatitis,	7-9
4. Leucoplacia oris,	9-10
5. Sprue. Stomatomycosis oidica,	10-14
Appendix: <i>a.</i> Leptothrix buccalis,	13
<i>b.</i> Stomatomycosis sarcinica,	14
6. Salivation. Ptyalism,	14-17
Appendix: Diminution of salivary secretion,	17
7. Fibrinous inflammation of the excretory ducts of the salivary glands. Sialodochitis fibrinosa,	17
Appendix: Mechanical retention of saliva,	17
8. Catarrhal inflammation of the soft palate and mucous membrane of the pharynx. Angina and catarrhal pharyngitis,	17-22
9. Chronic catarrh of the soft palate and pharynx,	22-25
10. Mycosis pharyngis leptothricia,	25-26

PART II.

DISEASES OF THE ŒSOPHAGUS,	26-50
1. Stenosis of the œsophagus,	26-33
2. Dilatation of the œsophagus,	33-37
3. Catarrhal inflammation of the œsophagus,	37-39
4. Phlegmonous inflammation of the œsophagus,	39-40
5. Corrosive inflammation of the œsophagus,	40-41
6. Round ulcer of the œsophagus,	41
7. Cancer of the œsophagus,	41-44
8. Hemorrhages from the œsophagus,	44-45
9. Perforation of the œsophagus,	45-47
10. Spontaneous rupture of the œsophagus,	47-48
11. Softening of the œsophagus. Œsophagomalacia,	48
12. Sprue in the œsophagus. Œsophagomycosis oidica,	49
13. Paralysis of the œsophagus,	49-50
14. Spasm of the œsophagus. Œsophagismus,	50-51

PART III.

	PAGE
DISEASES OF THE STOMACH,	51-105
A. Gastric affections associated with anatomical lesions,	51-100
1. Hemorrhage of the stomach,	51-56
2. Acute gastric catarrh. Acute gastritis,	56-59
3. Chronic gastritis,	59-64
4. Purulent gastritis. Gastritis phlegmonosa,	64-66
5. Toxic gastritis. Gastritis venenata,	66-67
6. Round ulcer of the stomach,	67-75
Appendix: a. Hemorrhagic erosions,	75
b. Follicular ulcers,	75
7. Cancer of the stomach,	75-82
Appendix: a. Gastric polypi,	82
b. Sarcoma,	83
8. Dilatation of the stomach. Gastroectasia,	83-94
9. Incontinence of the pylorus,	94-95
10. Degenerative changes in the stomach,	95
11. Atrophic changes in the stomach,	95
12. Softening of the stomach. Gastromalacia,	95-96
13. Rupture of the stomach. Gastrorhexis,	96-97
14. Animal and vegetable parasites in the stomach,	97-98
15. Foreign bodies in the stomach,	98
16. Changes in the shape and position of the stomach,	98-100
B. Functional Diseases of the stomach. Neuroses of the stomach,	100-105
1. Rumination. Merycismus,	100
2. Peristaltic restlessness of the stomach,	101
3. Hypersecretion of the gastric mucous membrane,	101
4. Nervous pain in the stomach. Gastralgia,	101-104
5. Nervous dyspepsia. Neurasthenia gastrica,	104-105
Appendix: Periodical vomiting,	105
Nervous gastroxyntsis,	105

PART IV.

DISEASES OF THE INTESTINES,	105-171
1. Acute intestinal catarrh,	105-112
Appendix: a. Cholera morbus,	112-114
b. Acute gastro-intestinal catarrh of infants,	114-118
2. Chronic intestinal catarrh,	118-123
Appendix: Phlegmonous enteritis,	123
3. Inflammation of the cæcum and vermiform appendix and surrounding parts. Typhlitis, perityphlitis, and paratyphlitis,	123-127
4. Stenosis and occlusion of the intestines. Entero-stenosis and ileus,	127-136
5. Round ulcer of the duodenum,	136-137
6. Intestinal cancer,	137-140
Appendix: a. Polypi,	141
b. Lipoma,	141
c. Angioma, myoma, sarcoma,	141
7. Animal parasites of the intestines. Helminthiasis,	141
Protozoa,	141-143
a. <i>Amoeba coli</i> ,	142
b. <i>Cercomonas intestinalis</i> ,	143
c. <i>Trichomonas intestinalis</i> ,	143
d. <i>Balantidium coli</i> ,	143
Flat worms. Platyodes,	143-151
a. <i>Bothriocephalus latus</i> ,	145-146
b. <i>Tænia solium</i> ,	146
c. <i>Tænia saginata</i> ,	146-147
Appendix: a. <i>Tænia nana</i> ,	151
b. <i>Tænia flavo-punctata</i> ,	151
c. <i>Tænia cucumerina</i> ,	151
d. <i>Tænia madagascariensis</i> ,	151
e. <i>Bothriocephalus cordatus</i> ,	151

	PAGE
Round worms. Nematodes,	152-159
Ascaris lumbricoides,	152-155
Appendix: Ascaris mystax,	155
Oxyuris vermicularis,	155-157
Tricocephalus dispar,	157-158
Anchylostomum duodenale,	158
Anguillula intestinalis and stercoralis,	158-159
Appendix: Trematodes,	159
8. Intestinal hemorrhage. Enterorrhagia,	159-163
Appendix: Melæna neonatorum,	163-164
9. Hæmorrhoids. Phlebectasia hæmorrhoidalis,	164-168
10. Nervous intestinal pain. Enteralgia,	168-169
Appendix: 1. Nervous constipation,	170
Nervous diarrhoea,	170
2. Embolism of the mesenteric artery,	170

PART V.

DISEASES OF THE LIVER,	171-228
A. Diseases of the biliary passages,	171-191
1. Stenosis and occlusion of the bile-ducts,	171-178
2. Catarrh of the bile-ducts. Cholangitis et cholecystitis catarrhalis,	178-181
3. Purulent inflammation of the bile-ducts,	181
4. Gall-stones. Cholelithiasis,	181-190
5. Parasites of the biliary passages,	190
a. Ascaris lumbricoides,	190
b. Echinococci,	190
c. Liver fluke. Distomum hepaticum,	190
6. Dropsy of the gall-bladder. Hydrops cystidis felleæ,	190-191
7. Empyema cystidis felleæ,	191
8. New growths in the biliary passages,	191
B. Diseases of the parenchyma of the liver,	191-225
1. Hyperæmia of the liver,	191-194
2. Perihepatitis,	194-195
3. Suppurative hepatitis. Abscess of the liver,	195-201
4. Chronic intestinal inflammation of the liver. Cirrhosis of the liver,	201-207
5. Acute yellow atrophy of the liver,	207-211
6. Fatty liver,	211-213
7. Waxy liver,	213-216
8. Cancer of the liver,	216-220
Appendix: a. Sarcoma,	220
b. Adenoma,	220
c. Fibroma, etc.,	220
9. Echinococcus of the liver,	220-224
10. Changes in the position of the liver,	224-225
11. Changes in the shape of the liver. Tight-laced fissure,	225
C. Diseases of the blood-vessels of the liver,	226-228
1. Stenosis and occlusion of the portal vein,	226-227
2. Suppurative inflammation of the portal vein,	227-228
3. Aneurism of the hepatic artery,	228

PART VI.

DISEASES OF THE PANCREAS,	229-230
1. Hemorrhage,	229
2. Pancreatitis,	229
3. Cancer,	229
Appendix: Diseases of the mesenteric and retroperitoneal glands,	230

PART VII

	PAGE
DISEASES OF THE PERITONEUM,	230-242
1. Inflammation of the peritoneum. Peritonitis,	230-237
2. Dropsy of the peritoneal cavity. Ascites,	237-242
3. Cancer of the peritoneum,	242
4. Parasites of the peritoneum,	242

SECTION IV.

DISEASES OF THE URINARY AND SEXUAL APPARATUS.

PART I.

SYMPTOMATICALLY IMPORTANT CHANGES IN THE URINE,	243-257
1. Albuminuria,	243-245
2. Hæmaturia,	245-252
3. Hæmoglobinuria,	252-254
4. Pyrocatechinuria,	254
5. Melanuria,	254
6. Chyluria,	254-255
7. Lipuria,	255
8. Fibrinuria,	255
9. Hydrothionuria,	255
10. Oxaluria,	256
11. Cystinuria,	257

PART II.

DISEASES OF THE RENAL PARENCHYMA,	258-308
1. Uræmia,	258-263
2. Ischæmia of the kidneys,	263-268
3. Passive congestion of the kidneys,	265-268
4. Bright's disease,	268-290
<i>a.</i> Acute diffuse nephritis,	268-276
<i>b.</i> Diffuse chronic parenchymatous nephritis,	276-281
<i>c.</i> Diffuse chronic interstitial nephritis,	281-290
5. Suppurative nephritis,	290-293
6. Paranephritis,	293-296
Appendix: Perinephritis,	296
7. Embolism and hemorrhagic infarction of the kidneys,	296-297
8. Waxy kidneys,	297-299
9. Cloudy swelling and fatty degeneration of the kidneys,	299-300
10. Cancer of the kidney,	300-303
Appendix: <i>a.</i> Sarcoma,	303
<i>b.</i> Adenoma,	303
<i>c.</i> Fibroma,	303
<i>d.</i> Cavernoma,	303
<i>e.</i> Lymphangioma,	304
11. Cystic kidneys,	304-305
<i>a.</i> Congenital cystic kidney,	304
<i>b.</i> Cyst-formation in chronic intestinal nephritis,	304
<i>c.</i> Other forms of cysts,	304
12. Echinococcus of the kidneys,	305-306
Appendix: <i>Pentastomum denticulatum</i> ,	306
<i>Cysticercus cellulosæ</i> ,	306
13. Movable kidney. Floating kidney,	306-307
Appendix: Abnormally low position of the kidney,	307
14. Anomalies in the shape and number of the kidneys,	307-308
<i>a.</i> Lobulated kidneys,	307
<i>b.</i> Horse-shoe kidneys,	307
<i>c.</i> Absence of a kidney,	308
<i>d.</i> Supernumerary kidneys,	308
Appendix: Aneurism of renal arteries,	308

PART III.

	PAGE
DISEASES OF THE RENAL PELVIS AND THE URETERS,	308-326
1. Hydronephrosis,	308-310
2. Pyelitis,	310-315
3. Renal calculi. Nephrolithiasis,	315-323
Appendix: Renal infarction,	323-324
4. Tumors in the pelvis of the kidney and ureters,	324
5. Parasites in the pelvis of the kidney and ureters,	325-326

PART IV.

DISEASES OF THE BLADDER,	326-345
A. Anatomically demonstrable diseases of the bladder,	326-340
1. Cystitis,	326-335
2. Cancer of the bladder,	335-338
Appendix: mucous polypi, lipoma, myxoma, etc.,	338
3. Parasites in the bladder,	338-339
4. Foreign bodies in the bladder,	339-340
B. Functional diseases (neuroses) of the bladder,	340-345
1. Nocturnal enuresis,	440-342
2. Hyperæsthesia of the bladder,	342
3. Spasm of the bladder,	342-343
4. Paralysis of the bladder,	343-345

PART V.

DISEASES OF THE MALE SEXUAL APPARATUS,	345-355
1. Male impotence,	345-346
2. Male sterility,	346-347
a. Aspermatism,	346
b. Azoospermia,	347
3. Spermatorrhœa,	347-351
a. True Spermatorrhœa,	347-350
b. Prostatorrhœa,	350
Appendix: Diseases of the suprarenal capsules,	351-355
Bronzed skin, Addison's disease,	351-355
Hemorrhage into the suprarenal capsules,	355



HANDBOOK OF PRACTICAL MEDICINE.

SECTION III.

DISEASES OF THE DIGESTIVE APPARATUS.

PART I.

DISEASES OF THE BUCCAL CAVITY, THE SALIVARY GLANDS, THE SOFT PALATE, AND THE PHARYNX.

1. *Catarrhal Inflammation of the Buccal Mucous Membrane— Catarrhal Stomatitis.*

1. **ETIOLOGY.**—Catarrh of the buccal mucous membrane may be primary or secondary, acute or chronic.

Primary stomatitis is generally the result of thermal, mechanical, or chemical irritants.

It is doubtful whether stomatitis alone may be the result of a general cold, but it may be produced by too cold, and especially by too hot articles of diet. Among the mechanical irritants may be mentioned the sharp edges of the teeth, which give rise to an inflammation of the mucous membrane of the tongue or cheeks. In infants, stomatitis is sometimes the result of violent sucking, particularly if the mother's breast does not contain much milk. It is sometimes produced by too early feeding with solid food. Continued talking and shouting are also said to produce stomatitis.

Among chemical causes may be mentioned the internal or local employment of preparations of iodine, bromine, arsenic, lead, mercury, and, according to Guipon, the prolonged administration of nitrate of silver. In some cases it is produced by mineral acids or other irritating substances and gases.

In drinkers and smokers it is the result of the irritation produced by alcohol and tobacco. It also occurs in infants whose mouth is not kept clean, and in which particles of milk are allowed to decompose. It is also produced occasionally by an irritating secretion from the nipple of the mother's breast. It may also be produced by vomiting, especially if the vomited matter is very acid. This is often most marked in dilatation of the stomach.

Secondary stomatitis is sometimes propagated from adjacent organs,

sometimes is associated with infectious diseases, or is the result of various disturbances of the general condition.

Stomatitis often accompanies dentition in children, and in adults is observed not infrequently in inflammation of the alveolar process, or with the cutting of the wisdom teeth. It may be associated with inflammation of the salivary glands, or of the mucous membrane of the nose, pharynx, or larynx. It is observed very often in gastric affections.

The affection may develop in all febrile conditions, especially in the infectious diseases (typhoid fever, measles, scarlatina, variola, erysipelas, phthisis, syphilis, etc.). It is sometimes communicated by the milk of cows or goats who are suffering from foot rot.

It is not infrequent in chlorosis, anæmia, and scurvy, and is observed occasionally as the result of stasis in diseases of the heart and lungs.

II. SYMPTOMS AND ANATOMICAL CHANGES.—In acute stomatitis there is usually, at first, a feeling of heat, dryness, and burning in the mouth. In nursing infants, these sensations are manifested by the frequent introduction of the finger into the mouth.

The inflamed mucous membrane is hot, and is often sensitive to the touch.

At first the buccal secretion is extremely scanty because the excretory ducts of the mucous follicles are occluded on account of the inflammatory swelling. At a later period, there is often a very profuse flow of saliva; in infants this often runs in a constant stream from the mouth, and produces erythematous inflammation of the integument of the chin. In adults the secretion flows backward and then passes into the stomach. The buccal fluid is usually acid, more rarely neutral, but, according to Bohn, it is never alkaline. As a rule, the secretion is at first tough, vitreous, transparent; later it becomes more fluid and cloudy, and is richer in cells. The salivation is explained in part by increased activity of the mucous follicles, but mainly by reflex stimulation of the salivary glands.

Perverse sensations of taste appear very early. Many patients complain of a flat, pasty taste, a sensation which may be resolved into several components, inasmuch as the tactile sensibility of the mucous membrane and the delicacy of taste are both impaired. Some patients complain of a bitter or foul taste, and the latter may be associated with a foul odor from the mouth. All these changes are the result, in part, of desquamation of the epithelium which accumulates within the buccal cavity and, mixed with débris of food and fungoid vegetations, undergoes decomposition.

The anatomical changes may be studied in detail in the living subject. The inflammation is more often circumscribed than diffuse. The inflamed parts are extremely red, either uniformly or in an aborescent manner. At the same time the mucous membrane is unusually swollen; this is particularly well marked if the cheeks or tongue are affected. The tongue then seems to be broader, and its edges show the indentations of the teeth. In some cases the mucous follicles take a more prominent part in the swelling, and this is recognized particularly on the mucous membrane of the lips and the border between the hard and soft palate. In these localities, we find small elevations, sometimes of a pearl-gray color, and upon pressure these often discharge a mucous or slightly puriform secretion. A red vascular areola is often seen around these nodules. In very rare cases, rupture of the follicles gives rise to superficial losses of substance.

Stomatitis is generally accompanied by very active loosening and

desquamation of the epithelium. The cells often accumulate on the inflamed parts and form white, smeary, partly removable patches, or a more diffuse deposit. It is found very often on the gums, inner surface of the lips, and on the tongue, upon which it forms grayish, yellowish, or brownish masses, or the color may depend on the character of the food. The swollen papillæ fungiformes sometimes project upon the surface of the tongue, and, as the epithelium of the papillæ is easily removed, numerous red prominences, hardly as large as the head of a pin, are found upon the otherwise coated tongue.

The coating of the mouth consists, in addition to débris of food, chiefly of pavement epithelium, often in large coherent masses; the cells contain fat granules. In addition, we find bacteria, particularly rod-shaped ones, pus-corpuscles (either scattered or in groups), occasionally red blood-globules and, according to Miquel, cholestearin crystals and

FIG. 1.



Coating of the mouth in chronic stomatitis, containing desquamated epithelium, threads of lepto-thrix, mucous and pus-corpuscles. Magnified 275 diameters.

lime salts (Fig. 1). Brown granular pigment is observed free or inclosed in epithelial cells.

As a rule, the general condition of the patient is very little affected. The bodily temperature is hardly at all elevated unless the primary disease is pyrexial. In infants, however, the temperature may rise considerably as the result of stomatitis, and general convulsions are observed occasionally, but it is doubtful whether they depend upon the stomatitis. There is often a feeling of increased thirst, and anorexia may be present, even though the stomach is unaffected. The foul taste and odor from the mouth occasionally give rise to nausea and even vomiting. As the ingestion of food increases the burning sensation in the mouth, infants sometimes refuse the breast for days, and emaciation and loss of strength are necessary consequences. The disease does not

often last more than a week. Chronic stomatitis either develops as such from the start, or follows relapsing acute attacks. The symptoms are similar to those of the acute form, except that they are less violent. After it has lasted for some time, permanent thickening of the submucosa sometimes follows as a result of the inflammatory hyperplasia.

III. DIAGNOSIS AND PROGNOSIS.—The diagnosis is easy, but we must be careful not to regard every coating on the tongue as evidence of stomatitis. A coating often forms during the night upon the posterior half of the tongue of perfectly healthy individuals, as the result of dryness and imperfect desquamation of the epithelium.

In the new-born a sort of physiological congestion of the buccal cavity develops during the first few days of life. This is produced by the irritation of the atmosphere, the movements of sucking and the ingesta, and should not be mistaken for stomatitis.

The prognosis as regards life is good, but serious symptoms sometimes develop in nursing infants. Complete recovery does not always occur; for example, in the case of drinkers and smokers.

IV. TREATMENT.—In addition to the removal of the cause the inflamed membrane must be treated locally. Washing the mouth with cold water often proves sufficient. The food should not be too hot, cold, hard, spiced, or irritating in other respects. Rinsing the mouth with chloride of potash (gr. lxxv. : $\frac{3}{4}$ vij., every two hours; especially after meals) is regarded, in some measure, as a specific. In little children a soft piece of linen is dipped into the solution and applied every two hours.

The following mouth-washes may also be mentioned: Salicylate of soda (3 ss. : $\frac{3}{4}$ iiiss.); carbolic acid (3 ss. : $\frac{3}{4}$ iiiss.); liq. alumin. acetic. (gr. lxxv. : $\frac{3}{4}$ iiiss., one tablespoonful in a cup of water), the latter especially if there is a disagreeable fœtor ex ore.

In chronic stomatitis applications of corrosive sublimate (gr. vij. : 3 xiv.) or nitrate of silver (gr. xv. : 3 vij.-xiv.) have been recommended.

2. *Ulcerative Stomatitis.*

(*Stomacace.*)

I. ETIOLOGY.—Inflammation, at first of the gums, then ulcerative degeneration and a foul stench from the mouth are the chief symptoms of the disease. It occurs sometimes sporadically, sometimes epidemically (especially in overcrowded and badly ventilated barracks, prisons, orphan asylums, and hospitals).

In the epidemics described by French military surgeons, the officers escaped almost entirely, and the privates suffered most, because the former were better fed and lived under more favorable hygienic condition.

Larrey reports that the disease broke out among Napoleon's troops after the battle of Eylau, probably from drinking snow-water.

Telluric and climatic influences are not unimportant. For example, the disease occurs with remarkable frequency on the coast of Holland and, according to Vauvray, in Port Said. The disease is most frequent in summer, and its outbreak is said to be specially favored by the occurrence of a hot spell after prolonged rains. It is more frequent in the cities than in the country, and particularly in the low, damp dwellings of the poor.

Anæmic and feeble individuals, convalescents, those suffering from phthisis, diabetes, scurvy, scrofula, and rachitis, are specially predis-

posed to the disease. Children manifest a marked predisposition to this as to most other diseases of the mouth.

Among one hundred and six cases observed by Bohn, eighty-four occurred in children. It is observed most frequently from the age of four to ten years.

The development of the disease depends upon the presence of teeth, so that infants prior to the period of teething, and toothless old people are entirely exempt. Bohn mentions an obstinate case of this disease which was cured by the extraction of the teeth.

The parasitic and infectious character of the disease has not been proven. There is no doubt that it often attacks several children of the same family, that it occurs epidemically in institutions, and not infrequently appears in separate houses at the same time, but it may be claimed that the same bad hygienic influences are acting upon all the affected individuals. But we must confess that to us the clinical history of the disease suggests an infectious disease.

As a rule, a direct cause cannot be demonstrated in the sporadic cases.

Toxic stomatitis is a special form of the disease. It is produced most frequently by the internal or external administration of mercury, but lead, phosphorus, and copper may act in the same way.

It develops so much more readily from the administration of mercury the larger the doses and the more rapidly administered. It hardly ever occurs in chronic mercurial poisoning because very small quantities are gradually absorbed. Certain individuals suffer from stomatitis after extremely small doses of the remedy. Children possess a certain degree of resistance to its influence, although calomel, which is so much used in children, is especially apt to produce stomatitis.

II. SYMPTOMS AND ANATOMICAL CHANGES.—The ordinary (non-toxic) form of the disease generally begins with a feeling of rawness and burning in the mouth, which is especially annoying on taking food.

The first objective signs always appear upon the gums. As a rule, the lower jaw is affected, often upon one side alone (particularly the left side). During the further course of the disease, the lower jaw continues to be more affected than the upper, and the latter may remain entirely unaffected.

Redness and swelling of the mucous membrane are first observed on the free edge of the gum, especially at the point of contact of two adjacent teeth. The free edge of the gum is loosened and swollen, and bleeds on slight contact. After one or two days, a yellow, smeary, pasty coating forms. This gradually increases in amount and if removed discloses an ulcer, usually with sharp edges and a grayish, speckled base. A large part of the gums may be converted gradually into a pasty, gray, or brownish necrotic pulp, which consists of epithelium, pus-corpuscles, red blood-globules, bacteria, and granular detritus. Bohn also observed a sort of algæ.

These changes generally appear first at one of the incisor or canine teeth, and gradually extend backwards or between the teeth to that portion of the gums which is adjacent to the tongue. The process may be interrupted by the absence of a tooth.

The same process is found occasionally upon the mucous membrane of the cheeks and the edges of the tongue. Careful examination shows that these ulcers are, to a certain extent, casts of others which are found upon corresponding parts of the external and internal surfaces of the gums—hence a sort of local infection. The cheeks and lips are not infre-

quently swollen. Secondary facial erysipelas may develop or, after recovery and cicatrization, the mucous membrane of the cheek, edge of the tongue, and gums may remain adherent to one another.

In addition, a nauseous fœtor ex ore is noticeable. The patients often infect the whole room within a few minutes.

As a rule, the neighboring lymphatic glands (submaxillary, submental, cervical) are enlarged, indurated, often tender on pressure and during mastication. The salivary glands are occasionally swollen and inflamed. They are almost always stimulated to increased secretion, and discolored, stinking, often bloody saliva runs almost uninterruptedly from the usually open mouth. The flow of saliva also continues during sleep, the fluid wets the pillow and the patient's sleep is disturbed, partly by the stench, partly by the discomfort arising from the wet pillow. The saliva may also flow backwards into the larynx and give rise to attacks of cough and suffocation.

The more actively the destruction of the gums progresses, the more the teeth are laid bare. They begin to loosen, and in severe cases may be readily removed with the fingers, without causing pain. The inflammatory process occasionally extends to the lower jaw, in which it produces inflammatory and necrotic changes.

The remainder of the buccal mucous membrane is often in a condition of catarrhal inflammation, and the tongue is coated, broadened, and its edges present indentations produced by the teeth.

The hard palate and the floor of the mouth rarely are affected. Under very unfavorable conditions, however, wide-spreading necrotic changes may develop in these parts and give rise to death after pyæmic symptoms (chills, high fever, loss of consciousness, meteorism, etc.). The process never extends to the pharynx.

In certain cases the patients have a very cachectic appearance, but, as a rule, the general condition is very little affected. There is usually very little or no fever, the chief complaints referring to the pain in the mouth, fœtor ex ore, perverse sensations of taste, antipathy to food and drink, and the annoyance caused by the salivation.

Ulcerative stomatitis may be acute or chronic. The acute form runs its course in one or two weeks, the chronic form lasts for months. The latter develops from the former if slowly healing ulcers are left over, or it appears as a chronic affection from the beginning.

Mercurial stomatitis is often preceded by peculiar sensations of taste, generally described as metallic by the patient. A sensation is felt as if the teeth were too long or loose; in addition, there is marked salivation. Finally, redness of the buccal mucous membrane, active desquamation of epithelium with its accumulation into smeary white masses upon the gums and lips, disagreeable fœtor ex ore, and finally ulcerative destruction (which may first affect the mucous membrane of the cheeks or tongue) become noticeable.

III. DIAGNOSIS AND PROGNOSIS.—The diagnosis is so easy that a mistake is hardly possible. The previous history will serve to distinguish ordinary stomatitis from the toxic form. The prognosis is almost always favorable, and an unfavorable termination is hardly ever observed except as the result of gross carelessness.

IV. TREATMENT.—We should first endeavor to remove the cause of the disease. Among local remedies, chlorate of potash occupies the first rank, and renders all other remedies unnecessary. The mouth should be washed with a solution of the potash salt gr. lxxv.: 3 vij. every two hours.

Vogel administers it internally, and holds that it is very rapidly excreted in the saliva. Feuvrier recommends that small crystals of the salt be taken into the mouth. If there are ulcers on one side, the patient should sleep upon the unaffected side in order to prevent the growth of the ulcer from prolonged pressure of the cheek against the gums.

Carbolic acid (gr. xxx.: $\frac{3}{4}$ iiiss.), permanganate of potash (gr. xv. : $\frac{7}{8}$ iiiss., one teaspoonful to a cup of water), liq. alumin. acet. (gr. lxxv. : $\frac{3}{4}$ iiiss., one tablespoonful to a cup of water), etc., have also been recommended. The ulcers may be cauterized with the solid stick. If children are unable to gargle, the chlorate of potash may be given internally, or applied with a brush. Special attention should be paid to the diet (meat broths, milk, eggs, diluted wine, beer).

3. *Aphthæ. Aphthous Stomatitis.*

I. ETIOLOGY.—Aphthous stomatitis is chiefly a disease of childhood, and is observed most frequently from the tenth to thirtieth months of life. In certain children, the cutting of each tooth is accompanied by an outbreak of aphthæ. It is much less frequent during the period of second dentition.

The disease is observed frequently among the children of the poor, who attach little weight to keeping the mouth clean. A decided predisposition is also manifested by anæmic, rachitic, and scrofulous children.

Inflammations and other irritations of the buccal mucous membrane may also produce aphthæ. This category includes catarrhal and ulcerative stomatitis, sprue, catarrhal and diphtheritic angina, sharp edges of the teeth, inveterate smoking, etc.

In certain cases, the development of the disease is associated with infectious diseases or certain local diseases. It occurs in scarlatina, measles, fibrinous pneumonia, typhoid fever, gastric, intestinal, and uterine affections. Aphthæ appear in some women during menstruation, in others during the puerperal condition or the period of lactation.

The affection develops not infrequently in an epidemic form, and contagious influences can occasionally be demonstrated. The epidemics appear most frequently in the summer, then in the autumn, particularly after severe changes in the weather.

Bohn denies the contagious character of aphthæ. Brief mention may be made of the following case: The servant of a teacher visited her family in a remote village in which an epidemic of aphthæ was prevailing. Two days later, the servant, who had returned to service, was taken sick with aphthous stomatitis, and, a few days later, the child under her care. Then two of the other children were affected, and ten days later other cases appeared in the village.

It has been claimed by various writers that aphthæ may be conveyed from animals to man.

II. SYMPTOMS AND ANATOMICAL CHANGES.—In nursing infants, aphthous stomatitis begins not infrequently with mild prodromal symptoms: slight fever, irritability, increased thirst, increased salivation, pain on nursing.

The characteristic changes depend upon the formation of round, white or yellowish patches, which are surrounded by a red zone, are slightly elevated, and cannot be removed from the mucous membrane. These patches may develop within a few hours.

They vary from the size of a pea to that of a lentil, in some places

are merely punctate. When they increase in size, they coalesce, and give rise to irregular, jagged patches, which are found particularly at the edges of the tongue and at the junction of the mucous membrane of the lips and gums. A considerable extent of mucous membrane may undergo the aphthous change.

If the development of aphthæ is associated with the eruption of the teeth, the white patches are found first or exclusively at the site of the new tooth. Under other conditions, the patches appear with special frequency at the tip of the tongue, its lower surface and edges, the mucous membrane of the lips, and border of the gums. They are also found on the hard palate, uvula, and tonsils.

According to certain authors, aphthæ sometimes occur upon the intestinal mucous membrane.

Fresh eruptions often appear after the outbreak of the original crop.

Aphthæ never form vesicles; they are the result of a fibrinous inflammatory process in the mucous membrane, the fibrinous exudation being deposited immediately beneath the epithelium of the mucous membrane. The white patches consist of finely granular fibrin, which contains a few round cells.

Recovery generally occurs from desquamation, more rarely from absorption of the exudation, but cicatrices are left over in all cases. If desquamation takes place, the epithelial cover first ruptures. The fibrinous exudation gradually loosens at the edge, and often rolls up. Finally, it is cast off entirely, and a shallow, at first congested portion remains, which is very rapidly covered with epithelium, and *restitutio ad integrum* is complete.

The disease usually lasts one to two weeks. It is serious only in weak, poorly-nourished children, because the movements of suckling and mastication cause pain, so that the little ones are unwilling to nurse, and their nutrition is impaired still more.

The aphthæ sometimes develop near the excretory ducts of the salivary glands, occlude one or the other duct (generally Wharton's duct), and give rise to tension, pain, and swelling of the gland on account of the stasis of saliva. If the duct is made permeable by a fine sound, a large amount of saliva is discharged, and the symptoms rapidly disappear.

Baillard and Bouchut have observed necrotic changes in a few cases. Relapses often occur in adults.

III. DIAGNOSIS.—The diagnosis is easy. The following mistakes are possible :

a. Clumps of casein which have remained in the mouth in little children. The red areola is absent, and the lump is readily removed.

b. Stomatitis is characterized by fœtor ex ore, a tendency to hemorrhage, and degeneration of the tissue of the mucous membrane.

c. Sprue is easily recognized by the characteristic fungus.

d. In herpes of the buccal mucous membrane vesicles are present, from which, upon puncture, a fluid exudes.

IV. TREATMENT.—Apart from the removal of the causes, treatment consists of the use of chlorate of potash, either as a gargle or by application with a brush, or internally (gr. xlv. : $\frac{3}{4}$ iiiss., a teaspoonful to table-spoonful every two hours).

Other remedies are unnecessary. The following have been recom-

mended : Brushing with muriatic acid, nitrate of silver, corrosive sublimate, borax, lime water, etc.

4. *Leucoplacia Oris.*

1. In leucoplacia oris we find whitish, gray, yellowish, or yellowish-gray patches, which are especially frequent on the tongue, but also appear upon the lips, mucous membrane of the cheeks, gums, hard palate, uvula, velum palati, and tonsils. Sometimes there are a few scattered plaques which hardly project above the level of the surrounding parts, sometimes the patches are distinctly elevated, especially at the edges, sometimes they form almost papillomatous excrescences. Occasionally they are almost horny in consistence, coalesce with one another, and cover a large part of the buccal cavity.

On microscopical examination, the epithelial cells are found to have undergone marked proliferation, the upper layers are loosened and swollen, the lower are not thoroughly developed. The papillæ of the mucous membrane appear flattened, the vessels are dilated, and the subepithelial tissue is infiltrated with numerous round cells.

The disease has been recently described under different names, such as pityriasis, ichthyosis, psoriasis, tylosis, teratosis linguæ et oris, lichenoid.

2. The causes are : irritation of the mucous membrane by tobacco, alcohol, defective teeth, gastric disturbances, gout, syphilis. It is often associated with diseases of the skin (psoriasis, lichen planus, eczema, ichthyosis, syphilides, and epithelioma of the lower lip). It is more frequent in men than in women (among twenty-six cases collected by Morris, twenty-two occurred in men, four in women). It is said to be remarkably frequent among the natives of India. It is rarely observed during childhood, and is usually observed between the ages of twenty and sixty years.

3. The symptoms sometimes consist merely of the visible changes in the mucous membrane. Some patients experience a feeling of rawness while eating. Impaired gustation and a hypochondriacal mood are occasionally observed, the latter being the result of the patient's belief that his stomach is constantly disordered on account of the supposed coating on the tongue. As a rule, the disease runs a chronic course (sometimes more than thirty years).

4. The diagnosis is easy, since sprue, aphthæ, and cauterization of the mucous membrane may be excluded by the history, acute course, and microscopical examination.

5. The prognosis is doubtful. Numerous cases have been reported in which the disease was followed by epithelial cancer of the tongue (thirty-one times among sixty-eight cases reported by Weir).

6. Existing causes of the disease should be removed. Carlsbad, Vichy, and similar waters may be employed, and, if necessary, antisyphilitic remedies. The following remedies have also been recommended: Arsenic (Fowler's solution, aq. amygdal. amar., $\text{āā} \frac{3}{4}$ ss., M. D. S. 5-10 drops t. i. d. after meals), hyoscyamus (aq. amygdal. amar., 3 v.; ext. hyoscyami, gtt. xx. M. D. S. 15 drops every two hours), or gargles of chlorate of potash, borax, salicylic acid, cauterization with chromic acid (one part to five) every three or four days. After excision of the patches, they often return in the cicatrix. The disease is often made worse by the use of iodide of potassium.

5. *Sprue. Stomatomycosis Oidica.*

I. ETIOLOGY.—Sprue in the buccal cavity depends upon the proliferation of a fungus, *oidium albicans* (probably *saccharomyces albicans* is more correct) upon the mucous membrane of the mouth.

It is observed most frequently in the new-born, especially from the second to eighth weeks of life. In adults it is generally the result of protracted exhausting diseases (phthisis, cancer, diabetes, leukæmia, typhoid fever, etc.).

In the new-born it is so much more apt to develop the more the general care of the patient, especially the cleansing of the mouth, is neglected. It is generally found in large maternity hospitals, foundling asylums, among the children of the poor, and in damp, poorly-ventilated houses. Feeble children, particularly those who are weakened by chronic diarrhœa, have a special predisposition to the disease, for if the movements of nursing and deglutition are not very strong, and the mouth is not kept very clean, particles of food are apt to remain in the buccal cavity. They here decompose, and afford a favorable soil for the proliferation of the fungus. The disease is more frequent among bottle-fed infants than among those nursed at the breast. The majority of cases occur during the summer.

Imperfect cleaning of the nipple of the bottle is often an exciting cause. Sprue is sometimes produced by placing the child at the breast of a nurse whose own child suffers from sprue, and whose nipple is not kept clean. The fungus has also been found in the air of the room in which there were children who suffered from the disease. Berg produced sprue by inoculation on the buccal mucous membrane.

I have recently observed a very peculiar case of this disease. The patient, a girl of 20 years, has always been pale and weak. For one and one-half years, the tongue has presented a diffuse, light yellowish-gray coating which is two millimetres thick, and can be easily removed. It consists of spurs and threads of *oidium albicans*. Salivation is so marked that the fluid is constantly running from the mouth. Sleep is disturbed on account of the salivation. The patient suffers from anorexia. Nothing abnormal can be discovered in the internal organs. The peculiar features of the case are: The age of the patient, the idiopathic origin of the disease, and the uselessness of all remedies which have been employed.

II. SYMPTOMS.—If the development of the fungus is slight, all symptoms may be absent. Profuse development of the fungus has a serious significance, both on account of the usually dangerous primary affection, and also on account of the danger produced by the sprue itself.

The tip and sides of the tongue are generally the first to be affected, then follow the mucous membrane of the cheeks, lips, gums, hard palate, *velum palati*, and uvula.

In severe cases, sprue is found in the pharynx, œsophagus, and upper part of the larynx. It is sometimes so abundant in the œsophagus as to give rise to occlusion of the canal. Cough and attacks of suffocation have been observed in sprue of the larynx.

Reubold showed that mucous membranes which are provided with cylindrical or ciliated epithelium offer a vigorous resistance to the proliferation of sprue. Only in exceptional cases is it found in the stomach, nose, or those parts of the air passages which are provided with ciliated epithelium. The fungus is sometimes found in the contents of the stomach

and intestines, but in such cases it has simply been swallowed. It may also be aspirated into the deeper air passages, and give rise to putrid bronchitis or pneumonia.

Sprue appears, at first, as a whitish or bluish-white, thin coating of the mucous membrane. On careful examination, it is found that, upon the tongue, it is situated mainly in the spaces between the fungiform papillæ and on the mucous membrane near the excretory ducts of the follicles. These punctate spots gradually increase in size and thickness, and larger grayish-white or yellow, later brownish or blackish patches are formed, which finally coalesce with one another.

At first, the patches cannot be removed by gentle rubbing, because they are situated beneath the uppermost layer of epithelium. At a later period, the epithelial layer bursts, the smooth surface becomes rough

FIG. 2.



Oidium albicans from the mouth of a child æt. 9 months. Magnified 275 times.

and uneven, and the deposit is removed, in part spontaneously, in part by mechanical means.

The deposit consists chiefly of spores and threads of the fungus (Fig. 2). The spores are round or oval, with a distinct contour, very refractile, sometimes uniformly finely granular, sometimes containing one or two larger nuclei. They may be scattered or collected into irregular masses, or arranged into so-called colonies. The thallus threads are elongated, straight, or slightly curved threads which vary in width. The broader ones contain transverse striations, while the narrower ones have a less distinct contour and hardly any transverse striæ. The width of the threads varies from 0.003–0.005 mm. The inside appears finely granular, and in places contains oval cavities. In many threads indentations are visible. Here or immediately beneath the transverse septa we not infrequently find spores sprouting out laterally; some of these

are converted into thallus threads. The same process may again take place from the latter.

In addition to the fungus we find desquamated cells of pavement epithelium and a few round cells. In some places the fungus has penetrated between the epithelium cells, or the threads are covered with individual epithelial cells. Rees states that the spores sometimes penetrate the epithelium cells and proliferate within the latter.

The development of sprue is not infrequently preceded by inflammatory changes in the mucous membrane. The latter is red, dry, hot, and sensitive to the touch. The infants often cry while nursing, and adults complain of burning and pain while eating. Afterwards salivation becomes profuse, and the mixed secretion of the mouth always has an acid reaction. Zweifel found that in the new-born the saliva contained no ptyalin.

Infants affected with sprue often suffer from profuse and obstinate diarrhœa, the cause of which is not always clear. The diarrhœa sometimes precedes the sprue, and favors the development of the latter by the exhaustion it produces; in other cases it is an accidental complication; and in a third series of cases it is assumed that the fungus is swallowed, produces decomposition of the ingested milk, and thus causes the diarrhœa.

The duration of the disease depends upon the care devoted to the patient. As a rule, it may be relieved in one or two weeks.

III. ANATOMICAL CHANGES.—The fungus enters between the upper horny layers of epithelium, proliferates in the middle layer of the epithelium, where it gives rise, as the result of compression, to disappearance of the cells with the exception of the nuclei, while the upper layers of epithelium are merely flattened. In a few places the fungus may pass into the submucous tissue, and may even enter the blood-vessels and lymphatics. The vessels are congested, but, as a rule, inflammatory changes are absent.

Zenker's case shows that it is possible that the fungus may be carried away by the current of blood, after it has entered the vessels, to the periphery; for example, to the brain, where it may act as an embolus and continue to proliferate.

According to Rees and Grawitz, the fungus of sprue is really identical with *saccharomyces mycoderma* which is often found upon alcoholic and many fermenting substances. At all events, it is not identical with *oidium lactis*, which produces the acid fermentation of milk.

The fungus is very widely diffused, and a slight preparation of the mucous membrane alone seems to be necessary in order to allow the proliferation in the buccal cavity of the numberless spores which are floating in the air. Haussmann states that sprue is found in the vaginae of eleven per cent of pregnant women, and thinks it is conveyed to the new-born during delivery. But this does not explain the striking fact that the new-born are usually free from the disease for the first few weeks.

IV. DIAGNOSIS.—With the aid of the microscope the diagnosis is easy. Patches similar to sprue are found when *sarcina* develops on the buccal mucous membrane, but this is distinguished by the shape of the fungus (vide Fig. 4, p. 14).

V. PROGNOSIS.—The prognosis is not always favorable. In some cases sprue is an unpleasant complication of a disease which is in itself dangerous, in others it may produce serious results by giving rise to dis-

turbance of nutrition, diarrhœa, occlusion of the œsophagus, or disease of the larynx, bronchi, and lungs.

VI. TREATMENT.—The prophylaxis is very important. In children the mouth should be cleaned after each meal, or after vomiting, and the bottle and its nipple should also be carefully cleaned and kept in water when not in use. After the child has nursed, the nipple of the mother's breast should be carefully washed. In adults, likewise, the mouth should be carefully cleaned after each meal.

If sprue has formed, the mouth should be washed with borax every two hours (℞ Sol. natri biborac., gr. lxxv.: ̄vij. D. S. Externally).

Chlorate of potash has no effect in this disease. The application of muriatic acid, nitrate of silver, chloride of iron, or alum is necessary

FIG. 3.



Leptothrix buccalis from the tartar of the teeth. Magnified 275 diameters.

only in very obstinate cases. Kehrer claims that weak solutions of borax favor the development of *oidium albicans*.

APPENDIX.

It is not surprising that the mouth, with its warm, moist atmosphere and its decomposing fragments of ingesta, should offer a favorable soil for the development of low organisms. A large number of mould fungi and bacteria may be found in every mouth, particularly in the coating of the tongue and the tartar of the teeth. We will here refer somewhat in detail to two varieties.

a. Leptothrix buccalis. This fungus—included by some among the algæ, by others among the schizomycetes—forms long threads, which usually project from a mass of granules and rods. The threads are sometimes uninterrupted, sometimes appear to be made of small divisions arranged like a rosary (Fig. 3). They are found abundantly in the tartar and, according to Leber and Rottenstein, play an important part in dental caries. On the application of iodine they assume a blue color. *Spirochæte plicatilis* is also found in the tartar.

b. Stomatomycosis sarcinica has been observed a number of times by Friedreich in patients who were enfeebled by chronic diseases. The fungus was sometimes present in such masses as to form small white patches. They are easily recognized by their square shape and characteristic grouping.

A black coating on the tongue has recently been described, in which fungus threads and spores were found by Lanceraux (*glossophyton* according to Dessoir). The disease is insignificant, but is occasionally obstinate.

A. Fraenkel has recently obtained bacteria, similar in appearance to pneumococci, from the mouth of healthy individuals. When pure cultures were made and inoculated upon rabbits, the animals died of septicæmia. Frænkel proposes for this bacterium the term coccus of

FIG. 4.



Stomatomycosis sarcinica. After Friedreich.

sputum-septicæmia. The comma-like bacillus of the mouth is not identical with Koch's comma-bacillus of Asiatic cholera.

6. Salivation. *Ptyalism*.

I. ETIOLOGY.—*Ptyalism* means increased secretion of saliva; as a general thing the saliva flows, in part, from the open mouth.

It occurs most frequently as the result of reflex irritation of the nerves of the salivary glands, and hence it is generally observed as a symptom of most diseases of the mouth (stomatitis, dental caries, dentition, inflammation of the maxilla, etc.). I recently treated a boy of ten years in whom the disease lasted three months after an attack of mumps. Both parotid glands secreted very profusely, especially the one which had been affected. The occurrence of salivation after the administration of mercury, more rarely of iodine, gold, silver, copper, lead, and arsenic, is probably the result of reflex irritation of the nerves of the salivary glands by preceding stomatitis. Substances which have a sharp taste (tobacco, condiments, etc.) may also give rise to increased secretion of saliva as the result of irritation of the buccal mucous membrane. *Ptyalism* may also be produced by trigeminal neuralgia.

In some cases, the reflex irritation is produced by remote organs. Frerichs produced *ptyalism* in dogs by irritation of the gastric mucous membrane. Clinically it is found that *ptyalism* is a frequent accompa-

niment of many gastric and intestinal diseases (gastric catarrh, ulcer and cancer, cardialgia, worms, etc.). It was formerly held that diseases of the pancreas, spleen, and liver gave rise to salivation, but this has not been proven.

Diseases of the genital organs (uterus and ovaries) not infrequently give rise to ptyalism. Stark reports two cases in which insane women suffered from salivation whenever they were affected with nymphomania. Men and women have complained to me not infrequently that they suffered from salivation immediately before and during coitus. Some women suffer from very annoying ptyalism during pregnancy.

The disease is sometimes the result of irritative conditions of the central nervous system. It is well known that certain gustatory sensations may produce ptyalism, and this is also true of violent emotions. Lépigne and Bochefontaine have shown that irritation of the anterior part of the cerebrum in dogs caused increased secretion of the submaxillary gland. Ptyalism is not infrequent in hypochondriacal, hysterical, and insane individuals. Paulicki reports the case of a melancholiac who suffered for five years from salivation, which occurred every other day from 8 A.M. to 9 P.M.; it was associated with diaphoresis and profound depression. The affection has been observed a number of times in diseases of the medulla and pons, which contain the first centre for the salivary nerves.

Certain vegetable substances may produce ptyalism by action on the nerve centres (jaborandi or its alkaloids, pilocarpine, physostigmine, nicotine, digitalis).

Sometimes it is the result of irritation of the peripheral nerves, especially diseases of the middle ear which affect the chorda tympani.

Some individuals suffer from ptyalism if they hear very high and shrill notes.

According to some writers, it is observed occasionally in certain infectious diseases (typhoid fever, intermittent fever, dysentery).

The cause of salivation sometimes remains unknown (idiopathic ptyalism).

II. SYMPTOMS.—The normal daily amount of saliva varies from two hundred to fifteen hundred ccm. In practice, the disease is very easily recognized if disturbances of deglutition (pseudo-salivation) can be excluded.

The patients state that saliva accumulates very quickly and in large amounts in the mouth. Speech is thereby interfered with, and the patients must often swallow the saliva, and finally are compelled to expectorate very frequently, or to allow the saliva to run from the open mouth. There is occasionally a feeling of tension in the region of the glands, and they may be found somewhat swollen and hard. Some patients also complain of a sensation of warmth in the glands.

On account of the various changes which may have been present in the mouth, the mixed buccal secretion does not always possess the same qualities. In cases of diseases of the mouth, the fluid is often acid, cloudy from profuse desquamation of epithelium, often foul-smelling and bloody or dirty in color.

The symptoms of ptyalism may be produced experimentally by the administration of jaborandi or pilocarpine (0.01 subcutaneously), and the secretion can be collected separately by the introduction of canulæ into the excretory ducts of the salivary glands. The saliva which first escapes has an alkaline reaction, after a while it becomes neutral or even

acid. The reaction often varies from time to time, but if the experiment is continued for any length of time, the saliva tends permanently to remain acid. This is also true of ptyalism which results from other causes.

The amount of fluid may be very considerable (as much as eleven thousand ccm. in twenty-four hours, according to some old reports). After a time, the fluid deposits a sediment, composed of epithelial cells, a few mucous and pus corpuscles, crystals of carbonate of lime, and amorphous albuminoid granules. In some cases, the saliva does not contain rhodankalium and ptyalin.

A clinical distinction should be made between transitory and continued ptyalism. The former lasts only a few hours or days, and presents a favorable prognosis, since it subsides upon the removal of the cause. Protracted ptyalism may persist for months, years, even for life, is often very unyielding under treatment, and may prove a source of danger.

The affection gives rise, as a rule, to disturbed sleep, either because the saliva is constantly flowing from the mouth, wetting the pillow, and thus rousing the patient very often, or because it flows into the larynx, and causes attacks of cough and suffocation. If the saliva flows into the pharynx, the patient will be disturbed by frequent acts of deglutition.

Erythema of the integument of the chin is produced occasionally by the overflowing saliva.

Digestive disturbances are often produced. The ingestion of the large amounts of saliva, mixed with air bubbles, gives rise to a feeling of fulness and distention in the region of the stomach, and interferes with gastric digestion. Constipation is generally, diarrhœa rarely, observed. The saliva accumulated in the stomach is occasionally vomited, especially in hard drinkers, who generally reject in the morning the saliva which has accumulated in the stomach over night. If the secretion is very profuse, diuresis may be scanty, or the relative amounts of urine and saliva vary on alternate days. In one of my cases, a slight degree of polyuria lasted for a number of days after the salivation was relieved. The patients may emaciate, their appearance becomes almost cachectic, and occasionally they die from exhaustion—less as the result of ptyalism than of the causes which have given rise to it.

III. DIAGNOSIS.—The disease is easily recognized, but we should never be satisfied with the diagnosis of ptyalism, since this is only a symptom of various diseases.

IV. PROGNOSIS.—This always depends upon the etiology, for while salivation is almost physiological in some cases (dentition of childhood), and in others may be relieved by removing slight causes (local diseases of the mouth), it may also constitute a very obstinate and serious disease (affections of the central nervous system).

V. TREATMENT.—As a matter of course, the causes should be removed whenever possible. If this cannot be done (for example, in diseases of the central nervous system), almost the only rational measure is the administration of sulphate of atropia, gr. $\frac{1}{4}$; pulv. althææ, q. s. ut ft. pil. No. xx. D. S. 1 pill t. i. d.; or atropiæ sulph., gr. $\frac{1}{4}$: 3 iij., $\frac{1}{2}$ to 1 syringeful subcutaneously. This remedy possesses an inhibitory influence upon the secretory nerves of the salivary glands. Very little can be expected from the use of astringent mouth washes, or the internal administration of astringents. The use of opium is recommended by many.

APPENDIX.

Little is known concerning diminution of the salivary secretion. It may be produced artificially by the administration of quinine. Patients complain not very infrequently of a peculiar dryness in the mouth. This is observed in diabetes mellitus and insipidus, and small contracted kidneys, *i. e.*, diseases which are associated with an abnormally profuse excretion of urine. Similar complaints are made in febrile diseases. Dryness of the mouth also appears occasionally as a symptom of old age, and in hysterical, hypochondriacal, and anæmic individuals. Buxton recently described diminished secretion of saliva after an attack of mumps, and, at the same time, impairment of taste.

Drinks should be offered frequently to febrile patients, and the lips and tongue smeared with vaseline. In a few other cases I have employed pilocarpine internally and subcutaneously, with temporary benefit. Buxton obtained relief from the application of one electrode to the back of the neck and the other to Steno's duct.

7. *Fibrinous Inflammation of the Excretory Duct of the Salivary Glands.*
Sialodochitis Fibrinosa.

Kussmaul was the first to call attention to this disease; it has been observed in Steno's duct and the duct of the submaxillary gland.

At first, retention of saliva results; the affected gland becomes painful, tender on pressure, and swollen. Fever, inflammatory phenomena, and erysipelatous changes in the skin were observed in one case, absent in two others. Pressure or catheterization of the duct discharged some purulent, flocculent, occasionally foul-smelling masses and fibrinous casts of the duct. In addition to fibrin and round cells, Weber found bacteria in the expressed plugs. In Kussmaul's case, the disease had lasted ten years, and was attributed to pressure. Relapses were frequent, and were preceded for a few days by a salty taste in the mouth.

Treatment consists in the removal of the plug in the duct by pressure or catheterization.

Appendix.—Mechanical retention of the saliva is sometimes the result of aphthous or diphtheritic changes near the opening of the excretory ducts, and which occlude these ducts. Occlusion of the canals may also be brought about by salivary calculi and foreign bodies. Verneuil thinks that this may also be the result of spasm of the muscular coat of the ducts. If the saliva is retained in the gland, the organ enlarges, the patient experiences a feeling of tension, and later of pain, and the integument appears tense and shining. These symptoms rapidly subside if the excretory duct is made permeable by the passage of a sound.

8. *Acute Catarrhal Inflammation of the Soft Palate and Mucous Membrane of the Pharynx.* *Angina, and Catarrhal Pharyngitis.*

I. ETIOLOGY.—This disease is generally accompanied by disturbance of deglutition. The act of swallowing is painful and produces the sensation as if the inflamed region were narrowed (hence the term angina). The anginal disturbances are partly the result of local narrowing from the swelling of the inflamed tissues, partly of hyperæsthesia of the mucous membrane, partly of paresis of the muscular tissue of the soft palate and pharynx from infiltration of the muscles with inflammatory

transudation. These disturbances are particularly severe when the soft palate is affected by the inflammation, because this part forms a natural narrowing of the tract.

In some cases, the soft palate and pharynx are affected simultaneously, in others only one or the other is inflamed, and finally circumscribed parts may alone be attacked. Hence the terms *angina et pharyngitis diffusa et circumscripta*.

The disease may be primary or idiopathic, secondary or symptomatic.

Primary angina and pharyngitis include the rheumatic, traumatic, thermic, and toxic forms.

Rheumatic angina not infrequently follows a wetting, when strong winds and changeable weather are prevailing, particularly in the spring and autumn. Nevertheless the question has been raised whether the term infectious angina should not be substituted for rheumatic angina. At all events, careful examination often shows that the supposed exposure cannot be proven to have been the cause of the disease. Furthermore, the local changes are not infrequently preceded for days by febrile prodromata—a feature which could hardly be expected in a local disease resulting from a cold. Furthermore, the general symptoms and local changes are often strikingly disproportionate to one another; in addition, the disease is found to occur in epidemics, and cases of infection are observed. The epidemic outbreak and infection are apt to occur in schools, barracks, hospitals, etc.

The bacterium of rheumatic angina has not yet been discovered. Nevertheless, we believe that a cold hardly acts in any other way than by favoring the development of bacteria.

The predisposition to infectious angina is very unequally distributed. Children are affected with special frequency, and distinct predisposition is manifested until the twentieth to twenty-fifth years. Feeble, anæmic, and scrofulous children are prone to the disease. Heredity occasionally plays a part in the etiology, and in some cases a predisposition is acquired. This is especially true of individuals who are improperly clothed, who enervate their constitution by avoiding cool baths and frictions, live in rooms which are excessively warm, and do not exercise in the open air. Why in one individual this organ, in another that one, constitutes the *locus minoris resistentiæ* is unknown.

Catarrh of the soft palate and pharynx may be produced in a mechanical way by foreign bodies, such as fish-bones and other sharp substances which have injured the mucous membrane or have been wedged into it. The inhalation of dust may have the same effect. The disease is also produced by too loud or continued speaking and singing (preachers, teachers, singers, officers, etc.).

Among the thermal irritants are excessively hot or cold articles of diet, the inhalation of hot vapors.

Chemical irritants may have a direct or indirect action. In the latter event, the toxic substance is first received into the blood-vessels and then is deposited upon the mucous membrane, where it gives rise to irritation. Direct action is observed after the ingestion of acids, alkalies, and other irritating substances; indirect action is observed after the internal administration of mercury, bromine, iodine, arsenic, antimony, gold, silver, lead and copper, belladonna and veratrine.

The secondary catarrhs of the soft palate and pharynx include inflammations which are propagated from adjacent parts, and those which occur during the course of certain infectious diseases.

The former variety is observed in stomatitis, inflammation of the nasal mucous membrane and larynx, and in gastric catarrh. The older physicians very often assumed the existence of a "gastric" angina, because coated tongue, vomiting, and anorexia often make their appearance in angina and pharyngitis. We must be careful, however, not to mistake the effect for the cause.

Among the infectious diseases, angina and pharyngitis are observed regularly in scarlatina; indeed, the angina may be the sole manifestation of scarlatina. Angina also occurs in variola, measles, and røtheln, and is often observed in typhoid, typhus, and relapsing fever, more rarely in fibrinous pneumonia. It is also observed in facial erysipelas, but we must here differentiate between a secondary angina and one which is propagated directly from the external skin to the mucous membrane. Inflammation of the soft palate and pharynx may occur as an independent erysipelas of the mucous membrane, independently of cutaneous erysipelas. The French have called attention to the relations between acute articular rheumatism and angina; the latter may either precede or follow the articular affection.

Syphilis very often gives rise to catarrhal angina and pharyngitis. Intermittent fever also exerts an influence in this direction, and a number of cases of intermittent angina have been described.

According to Niemeyer, visceral gout is sometimes manifested as a catarrhal angina (vide Vol. IV.).

II. SYMPTOMS AND ANATOMICAL CHANGES.—Acute catarrhal angina and pharyngitis not infrequently run the course of an acute infectious disease. The scene opens with a violent chill, followed by high fever (40° C. or more). After a while the patient complains of difficulty and pain in swallowing. A few days later, the fever subsides quite suddenly, not infrequently after an outbreak of perspiration, but a feeling of marked weakness persists for some little time. Epileptiform convulsions may occur in children at the time of the chill and high fever. The severity of the general symptoms often exhibits a striking disproportion to the local changes, a very slight circumscribed redness of the mucous membrane sometimes being associated with general symptoms of a serious character.

The symptoms just sketched occur particularly in so-called rheumatic angina; when it is the result of other causes, the disease often develops very gradually, and the subjective symptoms are more prominent.

The chief subjective symptom is the disturbance of deglutition. This is often particularly annoying on account of the increased production of saliva and mucus, which stimulates the patient to repeated deglutition. Occasionally there is a sort of tenesmus of deglutition. The more diffuse and intense the catarrh, and the greater the swelling of the inflamed parts, the more difficult and painful are the movements of deglutition. The pain is sometimes so great that the patient refuses food.

Speaking may also give rise to a feeling of tension and pain, on account of the traction on the structures of the soft palate. In cases in which the uvula is markedly inflamed, the enunciation of the letter *r* is especially impaired.

Spontaneous pains are often felt; they are located immediately behind the angle of the lower jaw, but sometimes radiate into the region of the ear.

The head is often held stiffly or obliquely, the latter when the inflam-

mation is unilateral; the head is then inclined towards the diseased side. Rotatory and nodding movements are followed by severe pain.

The jaws are sometimes almost immovable, a narrow fissure being left between them. Every active or passive effort to open the mouth gives rise to severe pain, and articulation is thus interfered with. If the tonsils are swollen, the speech becomes nasal in character. Saliva sometimes flows in an almost constant stream from the open mouth. If the disease is complicated with stomatitis, the saliva may be cloudy, even tinged with blood, and very foul smelling. A disagreeable *fœtor ex ore* is often observed.

The submaxillary glands near the angle of the lower jaw are usually enlarged and tender to the touch. Some authors state that the enlarged tonsils may be felt from the outside, but they seem to have been mistaken for swollen lymphatic glands. The movements of mastication are generally painful and difficult.

To examine the throat, the patient is placed in front of the window, the mouth opened as wide as possible, and the head directed upward so that the light falls full into the pharynx; the tongue is then depressed with a spoon or spatula. The view is generally improved if the patient says *a* or *ah*.

The examination is difficult in little children, and an assistant may be required to hold the little one and compress the nostrils. When the child opens his mouth to breathe, the spatula should at once be introduced.

The inflammatory changes have been divided into superficial, parenchymatous, and lacunar, although these forms may not be entirely distinct from one another.

In superficial catarrh, the inflamed mucous membrane is reddened and swollen. The redness is sometimes uniform and diffuse, sometimes in patches, sometimes hemorrhagic in places; its intensity varies greatly, and it may even have a bluish tinge. The swelling is especially marked in those places in which the submucous tissue is abundant and loose. The uvula may be thickened into a shapeless mass, and lengthened so that its tip rests on the base of the tongue, and gives rise to nausea and vomiting. The pillars of the palate may be swollen to such an extent that the tonsils appear very small in comparison. If the tonsils in particular are inflamed and swollen, they may almost touch one another in the median line. When one tonsil is alone affected, the uvula is pushed towards the opposite side. In the beginning, the secretion of the mucous membrane is often diminished, so that the inflamed parts appear very dry. At a later period, the secretion is increased, and the parts are covered with a vitreous or slightly puriform, cloudy mucus.

The follicles of the mucous membrane may be swollen. This is shown by the nodular appearance of the mucous membrane; the small, grayish granules corresponding to the enlarged follicles which are filled with secretion. The rupture of individual follicles gives rise to superficial losses of substance. Vigorous desquamation of the epithelium of the mucous membrane sometimes takes place. The swollen epithelial cells sometimes remain upon the mucous membrane as grayish deposits, and to the inexperienced eye may look like diphtheritic deposits. The inflamed parts also present a great tendency to hemorrhage, and this may be produced by contact during examination.

In parenchymatous (phlegmonous) angina, not alone the surface of the mucous membrane, but the tissue proper of the membrane or the in-

terlacunar connective tissue of the tonsils is involved. Hence the tendency to the formation of abscesses. Clinically, this form of the disease is characterized by very severe general symptoms. Anatomically it is characterized by considerable swelling of the inflamed parts. If the abscess opens spontaneously, there is danger of suffocation if the perforation occurs during sleep and the pus flows into the larynx. In some cases the pus perforates externally. Cases have been reported in which the carotid was eroded, and fatal hemorrhage ensued. In other cases, external fistulæ formed, with inflammation of the subcutaneous tissue of the neck and passage of pus into the thorax. Phlegmonous angina may also give rise to œdema of the glottis, which proves fatal unless we interfere quickly and actively.

The patients feel very much relieved immediately after spontaneous or artificial discharge of the pus. Suppuration generally continues two to four days, and then recovery follows. The pus sometimes has a nauseous odor.

The lacunar inflammation affects the tonsils alone. The border of the lacunæ is found to be very red, and at the same time the secretion of the follicles is increased. The lacunæ contain a thick, puriform, later caseous secretion, which varies from the size of a pin's head to that of a pea, has an extremely foul odor when squeezed, and is composed chiefly of rod-shaped bacteria, drops of fat, needles of the fatty acids, cholesterol crystals, and a few lymph-corpuscles.

Complications occur not infrequently in catarrhal angina and pharyngitis. If the tonsils are very much swollen, the naso-pharyngeal space may be narrowed to such an extent that respiration is possible through the mouth alone. But if the tonsils are almost in contact, the slit for the current of air may be too narrow, the patients suffer from dyspnoea and danger of suffocation, and cyanosis and signs of cerebral congestion make their appearance.

These symptoms are especially dangerous in infants at the breast, because they render nursing difficult or impossible. Pulmonary congestion, bronchitis, and pneumonia are apt to develop because the infants are unable to breathe well with the mouth.

Many patients complain of impaired hearing and ringing in the ears. This is sometimes the result of mechanical narrowing of the opening of the Eustachian tube, sometimes of catarrh of the mucous membrane of the tube. The latter may lead to otitis interna, and thus give rise to serious symptoms and dangers.

Gastric symptoms (vomiting, anorexia, constipation) occur very often in the course of catarrhal angina, and not infrequently the disease begins with such symptoms.

I have repeatedly found albumin and casts in the urine; hæmaturia has also been observed. Cerebral symptoms (convulsions, delirium, clouded consciousness) are especially frequent in children.

The disease generally lasts only a few days. Many so-called ephemeral fevers of childhood are the result of catarrhal angina. The disease rarely lasts more than one to two weeks.

One attack predisposes the patient to a relapse. It is sometimes found that the relapses chiefly or exclusively affect the former site of disease. Furthermore, the relapses often present the same character of inflammation (superficial, phlegmonous, lacunar) as the original attack.

The following sequelæ may occur : transition into chronic inflamma-

tion, hyperplasia of the tonsils, in very rare cases paralysis of the pharynx or general paralysis.

III. DIAGNOSIS AND PROGNOSIS.—The diagnosis can be made at once from a mere inspection of the parts. The prognosis, as regards life, is almost always good. Danger arises only in children if there is an unusually high fever. The prognosis as regards permanent recovery is not so good, since relapses occur repeatedly in many cases.

IV. TREATMENT.—Prophylaxis is an important part of treatment. If the patients have been coddled and enervated, the constitution should be gradually hardened by cold frictions and exercise in the open air, and by salt-water baths in summer. Individuals who are required to strain their vocal organs should spare them as much as possible. The inhalation of dust should be prevented by suitable apparatus, etc.

The treatment of acute angina and pharyngitis is causal, local, and symptomatic.

The causal treatment purposes a speedy removal of the causes (iodine and mercury in syphilis, quinine in intermittent fever, etc.).

In local treatment, inhalations and applications with the brush seem to us to be more suitable than gargles. We may employ solutions of chlorate of potash (gr. lxxv. : $\bar{3}$ vij.), alum, muriate of ammonia, bicarbonate of soda, etc.

Abortive treatment by the application of a strong solution of nitrate of silver (gr. xv. : $\bar{3}$ ss.-i.) is not always successful. Compression may be applied to the throat in the following manner: a cloth dipped in tepid water is wrung out and placed around the neck; it is then covered with a dry cloth, and finally with a piece of oiled silk.

If an abscess forms, the pus should be removed as early and freely as possible with the aid of the knife.

In relieving fever, preference should be given to antipyrin (3 i.-ij. to $\bar{3}$ xiv. of water by enema). B. Fraenkel obtained very rapid success from the use of quinine (gr. x.-xv.) in lacunar angina.

9. *Chronic Catarrh of the Soft Palate and Pharynx.*

Chronic Catarrhal Angina and Pharyngitis.

I. ETIOLOGY.—Chronic catarrh of the soft palate and pharynx is a frequent and annoying complaint, and not infrequently interferes seriously with the patient's ability to work. The inflammation may be diffuse or limited to a few parts of the palate and pharynx.

The disease is observed most frequently from the twentieth to the thirty-fifth years; it is rare in childhood. Men are more frequently affected than women. A feeble, nervous constitution predisposes to the malady. Hereditary influence is said to have been observed in a number of instances. Mackenzie and Lemon maintain that a very large pharynx predisposes to the disease.

Chronic catarrh sometimes develops as the result of repeated relapses of an acute attack. In other cases, the disease appears in a chronic form from the beginning.

All those causes which have been mentioned in the previous section may also act as the etiological factors in that form of chronic catarrh which follows repeated acute attacks. The following are the causes of that variety which is chronic from the beginning:

a. Constant and loud talking and singing; the disease is often observed in teachers, preachers, singers, actors, etc.

b. Immoderate use of alcohol is not an infrequent cause. The less diluted the alcohol, the more apt is the disease to develop.

c. Inveterate smoking, especially cigarette smoking, is also an etiological factor. It must be remembered, however, that a number of causes act together in some of these cases.

d. Inhalation of dust has also been mentioned as a cause, but Mackenzie thinks that its efficacy has been overestimated. This statement does not agree with our own experience.

e. The disease is sometimes observed as the result of chronic stasis in chronic diseases of the heart and lungs.

f. It is sometimes the result of other chronic diseases, viz., Bright's disease, malaria, syphilis, phthisis, scrofula, gout, chronic rheumatism of the muscles and joints, etc.

g. In some cases, the disease is propagated from other parts (catarrh of the nose, larynx, stomach, œsophagus).

h. According to certain authors, the disease may develop in a reflex manner (?), especially as the result of uterine diseases.

II. SYMPTOMS AND ANATOMICAL CHANGES.—The majority of patients who suffer from diffuse catarrh of the soft palate and pharynx are annoyed by a feeling of dryness, tickling, and a feeling as if a foreign body were present in the throat. This is particularly noticeable on awaking in the morning and, as a rule, is so marked in talking and singing as to seriously interfere with these acts. At the same time, the chronic laryngitis, which is generally produced by the same causes which have given rise to the pharyngitis, interferes with the clearness and timbre of the voice.

An unusual secretion of mucus often compels the patient to hawk and cough a good deal. If the mucus is very tough, movements of strangling and vomiting are not infrequently produced during expectoration. In many patients, these symptoms are especially severe in the morning after rising. Others attempt to get rid of the mucus by swallowing it.

The tonsils may be very considerably enlarged as the result of chronic inflammation. The speech then assumes an unpleasant nasal character, the patients are often forced to breathe through the mouth, many suffer from dyspnoea during sleep, and are disturbed several times a night. On account of the narrowness of the opening of the Eustachian tube, the patients often complain of difficulty of hearing and ringing in the ears—symptoms which may also result from propagation of the catarrh of the pharynx to the Eustachian tube without marked affection of the tonsils.

The uvula may also undergo hyperplasia, often projects upon the base of the tongue, and thus gives rise to strangling, nausea and vomiting. Tonsillar hypertrophy and hyperplasia sometimes constitute an independent affection, the result of circumscribed chronic inflammation.

Among other forms of circumscribed catarrhs, Schmidt makes special mention of chronic lateral pharyngitis; it often produces very annoying symptoms, and is readily overlooked on account of its concealed position.

Chronic catarrh is also divided into the superficial, parenchymatous, and lacunar varieties.

In chronic superficial catarrh, the mucous membrane, as a rule, has a brownish-red or grayish-red color. Some of the vessels of the mucous membrane are occasionally found in a condition of varicose dilatation. The secretion of mucus is increased so that the patients are compelled to

hawk frequently, and are especially hindered in singing and speaking. The masses of mucus occasionally contain specks of blood, and for this reason the disease is often mistaken for pulmonary phthisis. The production of mucus is sometimes so considerable that the posterior wall of the pharynx shines as if coated with varnish. In other cases, the secretion dries into greenish-gray crusts, especially at night, when the patients breathe through the open mouth. During the day, the patients experience an annoying sensation of tickling, or as if a foreign body were situated in the upper part of the pharynx and, after vigorous strangling movements, the crusts are expectorated.

The mucous membrane not infrequently appears uneven and granular (pharyngitis granulosa). The individual elevations generally have a grayish color, the furrows between them appear congested. The mucous membrane is sometimes extremely dry so that the patients are annoyed by a scratching, burning feeling; sometimes it is coated with tough, vitreous, or puriform mucus which necessitates frequent hawking and swallowing. These changes occur earliest, and to the most marked extent, upon the posterior surface of the pharynx.

In very many cases, pharyngitis granulosa is a follicular affection, *i. e.*, there is a chronic inflammatory hyperplasia of the follicular apparatus of the mucous membrane. Saalfeld has recently described proliferation of the lymphoid tissue of the mucosa around the excretory ducts, hypertrophy of the mucous glands, dilatation of the duct within the region of swollen tissue, so that the slit-shaped opening is seen at the apex of each granule; the mucous membrane around the granules is either unchanged or is thickened and infiltrated with cells.

Chronic parenchymatous catarrh includes the hyperplasiæ, especially of the tonsils and uvula, which have been previously discussed.

In chronic lacunar catarrh, yellow, stinking plugs—thickened products of inflammation—are found in the lacunæ of the tonsils. The plugs are occasionally calcified, forming tonsillar calculi which sometimes attain the dimensions of a pea. These plugs may be hawked up by the patient. Anxious individuals may regard the yellow masses as tubercles which have been expectorated from the lungs. A single lacuna may be alone affected, but this may be the starting-point of frequent acute, inflammatory exacerbations. The disease sometimes gives rise to intolerable *fœtor ex ore*. The patients themselves may perceive the foul odor, and I have treated a number of patients who suffered in consequence from an antipathy to food, and from gastric and intestinal catarrh, all of these symptoms rapidly disappearing after the primary affection was relieved.

Among the complications and sequelæ, bronchial asthma merits special mention (vide Vol. I., page 228). According to Rauchfuss, pharyngitis granulosa in children sometimes maintains a tendency to pseudo-croup (vide Vol. I., page 180). Individuals who are affected by chronic angina often suffer from acute and febrile exacerbations, and also exhibit a tendency to diphtheria.

The disease often lasts for years, sometimes for an entire lifetime, if the etiological factors cannot be removed (teachers, clergymen, smokers, etc.).

III. DIAGNOSIS AND PROGNOSIS.—The diagnosis is so easy that further remarks are unnecessary.

The prognosis is not grave so far as regards danger to life, but the disease is not very amenable to treatment, and has a tendency to relapse. Hope of permanent relief can hardly be entertained in the case of those.

individuals whose occupation renders the removal of the causes impossible.

IV. TREATMENT.—The causal treatment should occupy the first rank. Teachers, clergymen, etc., should abstain from the use of the voice as much as possible.

Local treatment should also be taken into consideration. Simple superficial catarrh may be treated by the methods described on page 4. Good results are sometimes obtained from brushing the pharynx with tinct. iodi., tinct. gallarum, equal parts. The applications should be made daily, and immediately followed by gargling with water. The treatment is discontinued for a few days if pains and violent inflammation develop.

This plan of treatment may also be adopted in pharyngitis granulosa. Application of liq. ferri sesquichlorat. (3 iss.: 3 i.), zinc. chlorat. (3 ss.: 3 i.), iodoform., acid. tannic., aa gr. xxvij., spirit. vin. dil. 3 xiv.

FIG. 5.



Threads of leptothrix and epithelium from the deposits in mycosis pharyngis leptothrifica. Iodine-preparation. After Hering. Magnified 630 diameters.

have been recommended. Some authors prefer the application of powders, for example, alum or tannic acid.

In advanced cases, each individual granule should be destroyed with caustics, such as nitrate of silver, chromic acid, or sulphate of copper. The galvano-cautery has also been employed with excellent results.

Surgical interference is the best treatment for hypertrophy of the tonsils and uvula.

10. *Mycosis Pharyngis Leptothrifica.*

1. This disease gives rise to the formation of yellowish patches (occasionally looking like horn), which are situated mainly in the crypts of the tonsils and base of the tongue. Inflammatory changes are absent, and there may be no subjective symptoms. Some patients complain of tickling, a feeling of a foreign body, and dryness in the throat.

The changes are often discovered accidentally. The disease is entirely benignant, though very obstinate. Only fourteen cases have been hitherto observed.

2. If small particles of the deposits are treated with a five-per-cent solution of potash, and then teased, they are found to be composed, in part, of threads of *leptothrix* which turn blue on the addition of tincture of iodine (Fig. 5). Cœci in zoogleva masses are also present. It is doubtful whether similar deposits are produced by other fungi.

3. The prognosis should be made with caution, because the fungus is apt to return very rapidly if mechanically removed. One case was rapidly relieved by smoking. Very little effect has been obtained by direct applications, gargles, or cauterization. Galvano-cautery and tonsillectomy afforded at least temporary relief.

PART II.

DISEASES OF THE ŒSOPHAGUS.

1. *Stenosis of the Œsophagus.*

I. ETIOLOGY.—Stenoses of the œsophagus are the result of abnormal conditions within the lumen of the tube, of diseases of its walls, or of morbid changes in adjacent organs (intra-œsophageal, interstitial, and extra-œsophageal stenoses).

Intra-œsophageal stenosis is the result, in most cases, of the ingestion of foreign bodies or abnormally large boluses of food. But even large, hard foreign bodies may be present in the œsophagus, although symptoms of stenosis are entirely absent. Not a few cases have been reported in which foreign bodies in the œsophagus were found upon autopsy, although no symptoms were produced during life. *Oidium albicans* may proliferate upon the mucous membrane of the œsophagus to such an extent as to occlude completely the lumen of the canal. Polypi which project from the wall of the œsophagus into its lumen may be regarded as on the border-line between the interstitial and intra-œsophageal causes of stenosis.

Among the interstitial causes, the most frequent ones are cancer and cicatrices, the latter being generally produced by poisoning with acids or alkalis.

The cicatrices are always preceded by ulcerative changes in the wall of the organ. The toxic cicatrices are the most important on account of their frequency; in many cases they do not form until weeks after the poisoning. Stenosis has sometimes been observed after the ingestion of a too hot bolus, evidently as the result of ulceration (from the burn) of the œsophageal mucous membrane. Deglutition of a very hard and sharp morsel may act in the same way. The disease has also been observed a few times after small-pox, which is attended occasionally with the formation of pustules on the œsophageal mucous membrane. Ulcers resembling the round gastric ulcer have been found occasionally in the lowermost part of the œsophagus. These are probably the result of digestion of the mucous membrane by gastric juice which has passed into the œsophagus during an attack of vomiting (peptic ulcers), and, according to Debove, may terminate finally in cicatricial stenosis. (Œso-

phageal stenosis is sometimes the result of syphilitic ulcerations following destruction of gummata in the walls of the organ; the canal may also be narrowed by the projection of gummata into the lumen. In very rare cases cicatrices and stenosis are produced by tubercular ulcerations.

Abscesses in the walls of the Œsophagus may act in the same manner as tumors of this region. In these cases the symptoms of stenosis have been known to disappear suddenly after rupture of the abscess. It has been justly questioned whether stenosis may be produced by hypertrophy of the Œsophageal muscular tissue. But the stenosis is occasionally the result of a congenital defect of development. The lumen of a circumscribed portion of the tube is unusually small, though the walls may be almost entirely unaffected. In these cases, the patients have generally suffered since childhood from difficulty in deglutition, especially if the bolus of food is too large.

Those forms of stenosis which are the result of muscular spasm (spastic strictures) are associated with changes in the walls of the Œsophagus.

Extra-Œsophageal stenoses may be the result of numerous diseases, of which the following are the most important.

Enlargement of the thyroid gland, whether the result of struma or of a true neoplasm (particularly cancer) sometimes gives rise to stenosis. The lateral prolongations of the gland often surround and constrict the Œsophagus, and Huber reports a case in which the strumous degeneration affected only the lateral horns of the gland, but no swelling had been noticed anteriorly during life. The stenosis is also produced by enlargement of the cervical tracheo-bronchial and mediastinal glands, and by inflammation of the mediastinal and cervical cellular tissue. Gallard reports a case following a metastatic cancer in the connective tissue between the trachea and Œsophagus (primary cancer in the stomach). The stenosis is sometimes the result of vertebral changes: abscess in tuberculosis of the vertebræ, tumors, exostoses, and marked lordosis of the spine. It has also been observed as the result of dislocation of the hyoid bone and clavicle, excessive length of the styloid process and ossification of the stylo-hyoid ligaments.

Among the diseases of the respiratory apparatus, cancer of the lungs and pleura gives rise to Œsophageal stenosis with relative frequency. Stenosis has also been observed after inflammatory changes and thickening of the arytenoid and cricoid cartilages, though these really affect the lower end of the pharynx. Thickening and ossification of the cricoid cartilage has also been described as a cause of Œsophageal stenosis. This is also claimed with regard to enlargement of the thymus gland, but has not been proven with certainty. Van Swieten mentions stenosis as the result of retraction of the apex of the lung. In certain cases Œsophageal stenosis is produced by changes in the circulatory apparatus (extensive pericarditis, hypertrophy of the heart, aneurisms of the aorta, subclavian or carotid arteries).

It was formerly assumed that stenosis of the Œsophagus was produced by rhythmical filling of the right carotid in cases in which this vessel, contrary to the rule, took its origin from the aorta below the left subclavian and passed to the right side of the body either between the Œsophagus and spinal column or between the Œsophagus and trachea. Hyrtl held, however, that this was conceivable only when that portion of the vessel which passes transversely across the Œsophagus is in a condition of aneurismal dilatation. If this condition is not present, any changes which may be produced must be looked for in the circulatory organs. It

is evident that the act of deglutition will be able, by means of compression, to interrupt, temporarily, the circulation in the right carotid. This harmonizes with the fact that the right radial pulse has been known to disappear during the act of deglutition, that some patients complained, at this time, of palpitation, anxiety, and syncopal attacks, and that, upon autopsy, the portion of the right carotid, which was situated centrally from the Œsophagus, was in a condition of aneurismal dilatation.

Zenker's case presented analogous conditions. The patient, a very short man, died suddenly, and no cause of death could be ascertained. It was found that the aorta passed over the right bronchus and then passed between the Œsophagus and spinal column to the left side of the latter. Hence it is possible that temporary occlusion of the aorta occurred during the act of deglutition, and that this proved fatal by giving rise to sudden anæmia of the brain.

II. ANATOMICAL CHANGES.—Stenosis of the Œsophagus affects most frequently the lower third, next the region of the bifurcation of the trachea. As a rule, there is only one stenosis, more rarely two or more. The length of the stenosed portion varies considerably. Cases in which the entire organ is narrowed are rare, and occur generally after the action of caustic poisons. The degree of stenosis may be so marked that a fine sound is passed through with difficulty, and cases have been reported in which cicatricial structures had converted the entire Œsophagus into a solid connective-tissue strand. Annular stenoses are apt to give rise to symptoms at a very early period, while the effects of stenosis may be compensated for a long time if only a portion of the transverse section of the Œsophagus is affected, or if it is compressed upon one side alone. In the latter event, Œsophageal adhesions, and hence inability of the organ to move to one side, may be a source of great danger.

The Œsophagus is usually dilated and sometimes forms a diverticulum above the site of stenosis. The muscular coat is often hypertrophied and the mucous membrane in a condition of chronic catarrh. Below the stenosis the organ is usually collapsed and its walls atrophic, while the mucous membrane presents numerous longitudinal folds.

We will not describe the anatomical changes which constitute the causes of the stenosis.

III. SYMPTOMS.—The most prominent symptom is difficulty in deglutition (*dysphagia mechanica*). This develops suddenly (foreign bodies, cases of spastic stricture), or appears gradually and keeps on increasing for weeks and months (the latter is by far the more frequent).

At first the patients have a feeling as if a large solid bolus had stuck fast in a certain spot. Gross errors are made by the patients with regard to localization. They endeavor to prevent the disagreeable sensation by masticating the food very finely or mixing it with a large amount of saliva, or by taking a sip of water to wash it down. After a certain length of time, however, all these manœuvres fail. Some patients attempt to obtain relief by making repeated movements of deglutition after swallowing a morsel, by making certain rotatory and nodding movements with the head and spinal column, or by stroking the side of the neck with the fingers. Pain is always felt while the food is passing the site of stenosis. Sometimes the first morsel produces severe symptoms, but subsequent ones pass more easily.

If deglutition is not performed with sufficient caution, great anxiety and dyspnœa may occur after a number of morsels have been swallowed.

This is owing to the fact that the food accumulates above the stenosis, and distends the œsophagus so that the latter compresses the air passages situated in front of it. Perhaps it is partly the result of mechanical irritation of the recurrent nerves which are situated close to the œsophagus. These symptoms subside as soon as the food has been regurgitated or has passed into the stomach.

Regurgitation of solid food is also a frequent, almost constant symptom of œsophageal stenosis. If the stenosis is situated high up, regurgitation takes place very soon after ingestion. If it is situated lower down, and especially if a diverticulum has formed, hours may elapse before the food is regurgitated. The regurgitated food appears macerated and swollen, has a neutral reaction, and is mixed with pus-corpuscles and desquamated pavement epithelium, often with fungus spores and threads.

Loquet observed violent singultus in œsophageal stenosis, particularly when the stenosis was situated below the diaphragm.

If the stenosis continues to increase, the patients are compelled to restrict themselves to fluid nourishment, and finally, ingestion through the mouth becomes impossible.

Examination with the sound and auscultation afford remarkably valuable aid in the recognition of the disease.

In examination with the sound we find an obstruction, which is overcome with difficulty or not at all, at the site of stenosis. In the latter event, a sound must be chosen which is sufficiently small to be passed into the stomach. The situation of the stenosis can be determined by measuring the distance between the apex of the sound, where the obstruction is felt, and the point at which the sound corresponds to the row of teeth. The situation of the stenosis in the body is then found by applying the sound externally along the buccal cavity and spinal column, and partly from our knowledge of the various dimensions of the œsophagus.

Length of the œsophagus,	25 cm.
Distance from the teeth to the beginning of the œsophagus,	15 "
Length of the cervical portion of the œsophagus,	5 "
" " dorsal " " " "	17 "
Distance between the beginning of the œsophagus and the point at which it crosses the left bronchus,	8 "
Distance between the teeth and the point at which the œsophagus crosses the left bronchus,	23 "
Length of abdominal portion of the œsophagus,	3 "

The red English sounds are preferable to the French ones because they are more durable and less apt to break. The olive-tipped sounds should be selected. Mackenzie recommends that the transverse section of the sound should be oval, corresponding to the shape of the œsophagus. The anterior third of the instrument should be dipped in warm water for one to two minutes before introduction, in order to make it more pliable, and it should also be smeared with glycerin, albumen, butter.

A sound should not be introduced until we are certain that the constriction is not the result of an aneurism, since the latter may be perforated by the instrument and produce rapid death from hemorrhage. Nor should it be employed if acute inflammatory changes are present.

In introducing the sound, the patient should sit on a chair and brace himself firmly against its back; the head is slightly raised, the mouth

opened wide, and a cork placed between the teeth. The physician then bends the end of the sound slightly downwards. The patient is then directed to protrude the tongue slightly, the index finger of the left hand is introduced as far as the base of the tongue, and the sound, which is held like a pen by the right hand, is pushed in over the left index finger. At the first attempts, the patients often suffer from nausea or even vomiting,



FIG. 6.
Œsophageal sounds. *a*, English sound with olive shaped tip
b, French sound with sharp tip.

or they stop breathing. Much can be done by calmly reassuring them. If an obstacle is encountered, the sound should be held quietly for a few seconds, then withdrawn a little, and again pushed forwards. In some individuals the involuntary contraction of the œsophagus causes a temporary obstruction to the passage of the instrument. If the obstruction is permanent, we are justified in making a diagnosis of stenosis. Smaller sounds should then be employed until we succeed in passing the stricture. We should not be satisfied until the sound has passed into the stomach, since stenoses which are situated more deeply may otherwise be overlooked. If the stenosis is very marked, it may be impassable except to catgut filiform bougies. Whalebone sounds with removable olive-shaped ivory tips are not much employed at the present time.

Sainte-Marie and Ferrié have constructed special instruments to measure the length of the stenosis. Sainte-Marie placed upon the sound a compressible olive-shaped tip, while the posterior extremity terminated in a manometer, which was filled with a colored fluid down to the zero point. When the sound reached the stenosis, the manometer rose on account of the compression of the tip, and did not fall to zero until the obstruction was passed. Ferrié fastened to the tip of the sound a little sac of goldbeater's skin. The sac collapsed when the sound met the obstruction. The sound was then passed through the obstruction, and the sac was distended by blowing into the sound, which was then withdrawn. The beginning of the lower portion of the stenosis could be recognized by the obstruction felt in withdrawal. The length of the stricture is the difference in the lengths of the sound in the first and second positions.

If fluid is swallowed by the patient, and if, at the same time, we place the index finger upon the hyoid bone, in order to recognize the beginning of deglutition by the upward movement of the bone, the sound produced is only heard when the œsophagus is ausculted alongside the spine as far as the site of stenosis. Lower down the sound is entirely absent, or is heard only after a certain interval—after the fluid has gradually passed through the stricture.

Abnormal gurgling sounds, which sometimes last a few minutes, are heard not infrequently above the stenosis. This method of examination is especially valuable when no bougie is at our disposal, or when the presence of an aneurism is suspected.

The cervical portion of the oesophagus is ausculted on the left side alongside the larynx; from the lower border of the cricoid cartilage to the region of the clavicle. Pharyngeal sounds are heard more frequently in this region than good oesophageal sounds. The thoracic portion of the oesophagus is ausculted immediately to the left of the spinous processes, from the seventh cervical to the ninth dorsal vertebra. The stomach begins at the tenth, sometimes at the ninth dorsal vertebra.

Waldenburg successfully examined the organ (oesophagoscopy) by means of an arrangement of mirrors.

Ziemssen distended the oesophagus by means of an effervescent mixture, in order to determine, by means of auscultation, dilatation of the oesophagus above the stricture.

If the occlusion of the organ becomes complete, the condition of the patient is truly distressing. He emaciates to a skeleton, the abdomen is sunken, the bowels are constipated for many days, and the amount of urine diminishes to an extreme degree. In a case of sulphuric acid poisoning under my care, not more than 3.5 gm. of urea pro die were passed during the last week of life. The patients often lie for days more dead than alive. The respirations are not infrequently extremely slow and irregular, and we must be on our guard against mistaking this condition for death, especially since the heart sounds are unusually feeble and the pulse barely perceptible. In one case under my observation, respiratory intervals of one and a half minutes occurred during the last two days of life.

Complete occlusion of the stenosis sometimes occurs suddenly. According to Mosetig, this is apt to occur when the kernels of fruit which have been swallowed are deposited above the stenosis. The patients sometimes attribute sudden complete occlusion to external causes. A patient who was suffering from stenosis as the result of cancer, became unable to swallow anything a few hours after catching cold while helping to extinguish a fire. Sudden stenosis, or temporary complete occlusion has been observed in oesophageal cancer without any previous symptoms.

Exacerbation and improvement of the stenotic symptoms sometimes alternate with one another. According to Koenig, the symptoms of stenosis which occur in women suffering from goitre occasionally grow worse at the period of menstruation, when the goitre enlarges. Temporary improvement may occur in cancerous strictures if portions of the cancer break off and render the passage freer. A few striking cases of cicatricial stricture have been reported, in which sudden and permanent improvement occurred, even after the operation of gastrotomy had been contemplated.

In some cases, death results from perforation into the mediastinum or air passages, pneumonic changes, or gangrene of the lungs (the two latter produced by the passage into the larynx of ingested particles of food which had been regurgitated).

IV. DIAGNOSIS.—The diagnosis is usually made with facility from the subjective symptoms, the results of exploration with the sound, and auscultation. As a matter of course, we should also endeavor to ascer-

tain the cause of the stenosis. This depends upon the previous history of the other symptoms.

V. PROGNOSIS.—The prognosis depends in part upon the cause; for example, it is unfavorable in cancer and aneurism. It also depends upon the degree of stenosis.

VI. TREATMENT.—Treatment should be directed against the cause itself and against the stenosis as such. The former requires, according to circumstances, the use of antiphlogistics, antisyphilitics, absorbents, nervines, narcotics, and surgical interference.

The local treatment belongs properly to the domain of surgery. In cicatricial stricture, gradual dilatation with larger and larger sounds should be adopted. The sound should be introduced daily, every other day, or once or twice a week, according to the intensity of the irritative symptoms produced by the operation. It should be kept *in situ* for a few minutes to a few hours, and this plan of treatment should be continued for a long time. Good results are often obtained from the use of distensible bougies, which are allowed to remain in the œsophagus, and, when they grow thicker, mechanically dilate the organ. The employment of forcible rupture of the œsophagus and of internal œsophagotomy has been abandoned. Strictures of the cervical portion are sometimes incised from the outside (external œsophagotomy). In other cases, the œsophagus is opened from the outside below the stricture, and an œsophageal fistula is made in order to nourish the patient. Gastrotomy, *i. e.*, the making of a gastric fistula through which to nourish the patient, has been performed successfully in a number of instances. Of twenty-seven operations performed from 1876 to 1883, fourteen (fifty-two per cent) were successful (under antiseptic precautions).

In one case, Trendelenburg introduced into the gastric fistula of a boy a rubber tube which was provided above with a funnel-shaped attachment. The boy chewed his food with the teeth, then spat it into the funnel, and then passed it into the stomach with the aid of a spoon.

Careful sounding may also have a surprisingly good effect, and make the passage permeable for a few days in stenosis produced by cancer. Great care must be exercised, however, in order to prevent perforation.

In stenosis produced by thickening of the cricoid cartilage, Wernher has advised that the larynx be lifted forwards and upwards during the act of deglutition in order to render possible the entrance of the bolus into the œsophagus.

The character of the diet is very important in all forms of œsophageal stenosis. We may recommend milk, eggs to which salt, sugar, wine, liquor, coffee or tea has been added, beef-tea (mixed with an egg or milk), pigeons or chickens cooked into a soup which has been strained through a cloth, wine, beer, beer soup with egg, cocoa, chocolate. So long as pulpy substances can pass the site of obstruction, we should order scraped meat and ham, Leube-Rosenthal's meat solution, thin porridge of potatoes, peas, and lentils. It has recently been proposed to keep soft sounds permanently in the œsophagus in order to facilitate the passage of food into the stomach.

If the passage of food through the œsophagus is no longer possible, and œsophagotomy or gastrotomy is impracticable, we must endeavor to maintain life by artificial nourishment through the rectum.

This method is not capable of maintaining life permanently, for, in the most favorable cases, only one-fourth of the amount of albumen

necessary for nutrition is absorbed from the rectum. Eggs are absorbed only to a very slight extent; their absorption is increased by the addition of salt, but this gives rise to such irritation of the gut that the enemata are not retained. Beef juice and peptones are partly absorbed, but they are borne very poorly, and irritate the mucous membrane. Experiments have recently been made with injections of blood, but no positive results have yet been obtained. As a good mixture for nutritive enemata we can recommend meat soup mixed with an equal amount of milk and starch, but the soup should not be very salty; the injection should have the temperature of the body, and not exceed two hundred to three hundred cubic centimetres in amount. It is best to introduce it slowly through a rubber tube with a funnel attachment. Still better are Leube's meat-pancreas injections, which are made in the following manner: $\frac{3}{4}$ v. to x. meat are chopped fine, $\frac{3}{4}$ xiv. to $\frac{3}{4}$ iiiss. finely-chopped pancreas, which is free from fat, are added, and then mixed into a fine porridge with $\frac{3}{4}$ iiij. to ivss. lukewarm water. This mixture, at the temperature of the body, is then injected into the rectum. This mass is sometimes digested so thoroughly that the fæces are almost entirely normal in appearance. Mackenzie has recently recommended the following mixture: Boiled mutton or chicken, $\frac{3}{4}$ v.; milk, $\frac{3}{4}$ xiv.; fat, $\frac{3}{4}$ vi.; brandy, $\frac{3}{4}$ ss.; water, $\frac{3}{4}$ iiij. This is strained, rubbed into a porridge, and injected twice a day at a temperature of 35° C. Once a week, the rectum is cleared out about three to four hours before the injection of the nutritive enemata.

The attempt has been made to secure artificial nourishment by subcutaneous injections of oil, cod-liver oil, milk, beef juice, yolk of egg, defibrinated blood, and syrup.

2. Dilatation of the Œsophagus.

Dilatation of the Œsophagus may extend over the entire length of the organ (total dilatation), or only over small portions (partial dilatation). Sometimes the dilatation affects only a circumscribed portion of the transverse section of the organ (diverticulum). The latter variety is divided into pulsion and traction diverticula, according as the diverticulum is produced by a force from within or by traction from without. Dilatation of the Œsophagus sometimes develops as an independent disease, sometimes from previous stenosis (primary and secondary dilatation).

a. Primary total dilatation has been described as a congenital condition; this is, perhaps, the result of imperfect development of the muscular coat and congenital atony of the entire wall of the organ. In other cases, the condition is acquired in later years as the result of a blow on the chest, lifting a heavy load, the sticking fast of a hot bolus, ingestion of a large amount of hot water, perhaps chronic catarrh of the Œsophagus which has developed primarily or has been propagated from the stomach. Habitual vomiting has also been mentioned as a cause of this condition.

The dilatation is sometimes quite uniform and cylindrical, sometimes spindle-shaped, the greatest amount of dilatation being generally situated in the middle of the thoracic portion. The Œsophagus is occasionally elongated, and may be thrown into loops like the intestines. Luschka describes a case in which the organ was 46 cm. long (normal length 25 cm.), and 30 cm. in circumference (normal circumference 7.5).

The dilatation is sometimes so great that a man's arm can be intro-

duced into the organ. The mucous membrane is not infrequently in a condition of chronic catarrh, and occasionally presents superficial losses of substance; the epithelium may be very much thickened, almost warty. The muscular coat is often very thin in cases of congenital dilatation. In one case, Klebs found fatty degeneration of the muscular fibres, and Stern describes an infiltration of the mucosa and muscular coat with round cells. In other cases, the submucous and muscular coats have been found thickened.

As a rule, the first symptoms develop from the ages of fifteen to twenty years. The prominent symptoms are difficulty of deglutition and regurgitation of food. The patients may feel that the food does not reach the stomach. They have difficulty in swallowing, may take only small amounts of food at a time, and these must be moistened with large amounts of water; or they employ spoons and other instruments to push the food into the first part of the Œsophagus. Davy's patient could only swallow if he assumed a position midway between the dorsal and sitting position, and allowed the right arm to hang over the back of the chair.

Ingestion of food is sometimes followed by a sense of oppression, fear of suffocation, palpitation, and attacks of syncope—the result of pressure of the food which remains in the Œsophagus upon the heart, lungs, and adjacent nerves.

After a time, the food is regurgitated. It is often very little changed, in other cases it appears macerated. The regurgitated masses are usually alkaline or neutral in their reaction—a proof that they have not been in the stomach. The starchy ingredients of the food may be converted into sugar. The partly decomposing masses in the Œsophagus often give rise to a pestilential fœtor ex ore.

The disease often lasts five to ten years. Emaciation occurs so much more slowly the larger the amount of food which passes into the stomach, but death from starvation finally occurs. In Ogle's case, the fatal termination was accelerated by pressure of the dilated Œsophagus upon the thoracic duct.

The diagnosis depends upon the fact that, in persons who present the symptoms just described, the Œsophageal sound can not alone be passed into the stomach without obstruction, but that lateral movements of the sound enable us to recognize an unusually wide lumen. It must be remembered, however, that if the dilated Œsophagus is elongated and convoluted, the passage of the sound into the stomach may meet with great difficulty. Distention with carbonic acid may enable us to distinguish an unusually wide tympanitic zone along the spinal column. For this purpose, we may give the patient a teaspoonful of tartaric acid in half a wineglassful of water, then an equal quantity of bicarbonate of soda. It should also be remembered that distention of the Œsophagus with food will produce dulness on percussion along the spine, and that this disappears as soon as regurgitation has occurred.

The prognosis is unfavorable.

The treatment consists chiefly in the administration of nutritious fluid food, which should be given often, but in small quantities. Nourishment by means of the Œsophageal sound or enemata, or perhaps gastrotomy may become necessary under certain circumstances (vide page 32).

b. Primary partial dilatation of the Œsophagus is generally congenital.

This category includes the so-called "præstomach," which is a circumscribed dilatation immediately above the cardiac end of the stomach.

c. Secondary dilatation of the Œsophagus develops whenever a stenosis is present in any part of its course. The stasis of food above the constriction may give rise to gradual dilatation of the organ, but this occurs only in rare cases. It has also been stated that stenosis of the pylorus may produce secondary dilatation of the Œsophagus, particularly if the walls of the stomach are unyielding. Secondary dilatation is generally the result of acquired stenoses, rarely of congenital stenosis, especially if situated near the cardiac end.

The wall of the Œsophagus is thickened, occasionally the individual muscular strata are separated from one another, and in such places the wall is thinned. The anatomical changes mentioned in the remarks on primary dilatation are also noticeable.

The symptoms are generally concealed by those of Œsophageal stenosis. A diagnosis of dilatation secondary to a stenosis may be made if regurgitation occurs quite a while after the food is ingested, and if a sound can be moved freely to the sides above the constriction. The diagnosis may also be facilitated by distention of the Œsophagus with carbonic acid, or the appearance of dulness on percussion alongside the spinal column.

The prognosis depends upon the primary affection, and the treatment is the same as that of Œsophageal stenosis.

d. Pulsion diverticula are rarely observed. Almost all of them are situated at the transition of the pharynx into the Œsophagus, indeed they may be regarded as diverticula of the pharynx. As a rule they take their origin from the posterior wall of the organ, either in the median line or on one side. They form sacculated projections which at first press downwards between the spinal column and posterior wall of the Œsophagus, but later appear on one or both sides between the Œsophagus and larynx. Their size varies from that of a pea to that of a child's head. The lining mucous membrane is generally in a condition of chronic inflammation, is often thickened, and here and there presents epithelial losses of substance. According to Zenker and Ziemssen, the diverticulum possesses no muscular coat, but this is not true of all cases.

The disease is by far more frequent in men, and, as a rule, the first symptoms appear after the age of forty years. But this does not imply that the predisposition is not congenital. On the contrary, it appears to be generally the result of a circumscribed defective development of the muscular coat. In Feré's case, the muscular coat was absent in a circumscribed locality. Under such circumstances the food and foreign bodies will gradually dilate the Œsophagus and may produce a diverticulum at the congenitally weak spot. The following have been mentioned as the exciting causes of this form of the disease: ingestion of foreign bodies, injury in the region of the neck, wearing a tight collar, poisoning with alkalis. In some cases no cause can be discovered, and even those mentioned are not above criticism. Zenker and Ziemssen showed that the formation of these diverticula is favored by certain physiological changes. For example, calcification of the cricoid cartilage narrows the passage from the lower part of the pharynx to the upper part of the Œsophagus. As the muscular coat is very thin in the lower part of the pharynx, the stasis of food in this locality may produce gradual dilatation.

The symptoms develop very slowly. At first the food is retained in

the diverticulum only at intervals. As it increases in size it sometimes appears, after eating, as a tumor on one or both sides of the neck. Some patients can empty it by stroking the tumor. This manipulation may be attended by peculiar clucking sounds or, as in Betz's case, by a sound as if air were being pressed through a narrow opening. Similar sounds are sometimes audible during the ingestion of food, and are heard occasionally at a considerable distance.

The more the diverticulum is filled with food, the more its opening approximates the median line of the œsophagus, while at the same time the increasing dimensions of the part situated below the opening compress the œsophagus. If the diverticulum is sufficiently large, pressure upon the heart, trachea, bronchi, and recurrent nerves may give rise to marked dyspnœa, danger of suffocation, and palpitation of the heart. The food is regurgitated, partly in a macerated, partly in a decomposed condition, some time after it is ingested, and fœtor ex ore is often noticeable. The diverticulum sometimes receives the largest proportion of the food, so that the patients are exposed to the danger of death from starvation.

The diagnosis is based principally upon the appearance of a tumor in the neck after the ingestion of food, and the spontaneous development of murmurs while food is being swallowed. The tumor gives a dull or dull tympanitic percussion sound, and often disappears after stroking. Under certain circumstances the diagnosis may be rendered still more certain by cautious distention of the œsophagus with carbonic acid, if a tumor then appears which is tympanitic on percussion. It is especially characteristic of the formation of a diverticulum that, in examination with the sound, the instrument sometimes passes into the stomach, sometimes into the diverticulum. If two sounds are introduced, it is sometimes found that one enters the diverticulum, the other passes into the stomach. The more the diverticulum is filled with food, and accordingly the more its opening corresponds to the course of the œsophagus, the more likely it is that the sound will enter the diverticulum.

The prognosis is grave, inasmuch as the disease is not very amenable to treatment, though the patients often attain old age.

Proper nourishment, especially fluid food, is the first requisite in treatment. If the symptoms of impermeability of the canal become very marked, we may nourish the patient by the aid of the œsophageal sound. Finally, it may be necessary to perform gastrotomy. The textbooks on surgery should be consulted with regard to the removal of the diverticulum itself.

e. Traction diverticula are situated most frequently near the bifurcation of the trachea. They are usually associated with disease of the bronchial and tracheal lymphatic glands. As the result of periadenitic inflammation, an adhesion forms between the inflamed glands and the outer wall of the œsophagus. If the glands undergo retraction, the œsophagus experiences a local traction outwardly. The same effect may be produced by callous inflammation in the mediastinum, which has developed either spontaneously or as the result of tuberculosis of the spine, or pleurisy.

These diverticula are usually single, rarely multiple, and do not attain large dimensions. They involve either the anterior or lateral wall of the œsophagus, and their apex, which is usually funnel-shaped, may point upwards, downwards, or horizontally. Their depth is generally

two to eight mm., rarely twelve mm. The muscular coat is sometimes present, sometimes absent.

On account of the small size of the diverticula, disturbances of deglutition may not be produced. The chief danger arises from the tendency to perforation. This may be produced by sharp foreign bodies which have been caught in the diverticulum, by food which has decomposed in the same situation; occasionally the cause of perforation remains unexplained. The results of perforation are not always alike. In some cases, the Œsophagus is brought into communication with a cavity which is filled with a mass of softened lymphatic gland. Further perforation then occurs into a bronchus, and the softened masses are then expectorated, or are aspirated into the deeper air passages, and produce pneumonia or pulmonary gangrene. In the first event, recovery is conceivable. A communication with a pulmonary cavity, and the formation of a fistula, sometimes takes place, or purulent, ichorous pleurisy, pneumopericarditis, or pericarditis develops, or erosion of a large vessel (aorta, pulmonary artery).

Traction diverticula occur at all ages, and equally in both sexes. Their development can generally be traced to the period of childhood. As a rule, the patients are scrofulous, phthisical, or inhale a good deal of dust, since all these conditions are apt to produce inflammation of the tracheo-bronchial glands.

The condition is hardly recognizable during life, unless the signs of perforation occur, and all other causes may be excluded.

3. *Catarrhal Inflammation of the Œsophagus.*

I. **ETIOLOGY.**—Catarrhal inflammation of the Œsophagus is sometimes of mechanical, thermal, or chemical origin, sometimes it is propagated from adjacent parts, sometimes it is produced by constitutional diseases.

Thus acute catarrh may be produced by the ingestion of hard, sharp bodies, by the unskilful use of the Œsophageal sound, by the ingestion of too hot or too cold articles of food, of acids, alkalies, or other irritating substances. Œsophagitis is sometimes the result of frequent vomiting, especially if the vomited matters are very acid. Catarrh of the mucous membrane is often observed above the site of a stricture as the result of the violent irritation produced by the stagnant and decomposing food. Chronic Œsophageal catarrh is also produced in hard drinkers and inveterate smokers, in the latter as the result of the ingestion of saliva mixed with irritating tobacco juice.

It is sometimes found that the catarrh extends to the Œsophagus from the pharynx or stomach, and also from inflammations of the spine, mediastinum, pericardium, even of the larynx or bronchi.

Acute catarrhal Œsophagitis is often observed in acute infectious diseases (measles, scarlatina, typhoid fever, variola, cholera, diphtheria, etc.).

Chronic Œsophagitis is kept up not infrequently by chronic respiratory and circulatory diseases. It is also observed in syphilis and phthisis.

II. **ANATOMICAL CHANGES.**—The catarrh may be acute or chronic, circumscribed or diffuse.

Acute Œsophageal catarrh is characterized by loosening and profuse desquamation of the epithelium, which becomes opaque and whitish. Congestion is not prominent, because it is partly concealed by the condition of the epithelium cells. The mucous follicles are sometimes swollen.

They form small, gray, transparent elevations, situated generally in longitudinal rows upon the folds of the mucous membrane, and discharging upon pressure a mucous or muco-purulent fluid. They are often surrounded by an areola of injected vessels.

At the beginning of the catarrh, the mucous membrane is occasionally very dry, but later it is generally covered with an abnormally abundant mucous or muco-purulent secretion.

Very violent catarrh may give rise to superficial, but usually not very extensive losses of substance. These are produced sometimes by active desquamation of the epithelium, sometimes by shallow ulceration of the follicles, the latter being situated occasionally in longitudinal rows. Catarrhal erosions and ulcerations cicatrize, but do not often give rise to stenosis. In very rare cases, abscess and gangrene have been observed as complications and sequelæ.

It should be remembered that the mucous membrane of the new-born is physiologically congested, probably as the result of irritation produced by the food during the first few days of life. Some authors attribute it to changes in the foetal circulation.

In chronic œsophageal catarrh, the mucous membrane has a brownish-red or grayish-red color. The epithelium is thickened, the mucous membrane covered with a tough, vitreous, or puriform secretion. Superficial ulcers are often present.

Zenker and Ziemssen deny the occurrence of blackish or slate-colored pigmentation of the mucous membrane as the result of long-standing catarrh. One of the sequelæ is dilatation of the œsophagus, probably from relaxation and hypertrophy of the muscular coat. Zenker denies that this hypertrophy may become so excessive as to lead to stenosis of the canal.

In other cases, the mucous membrane becomes hypertrophic, and gives rise to the formation of polypoid or papillary proliferations. The hypertrophy sometimes extends to the peri-œsophageal cellular tissue.

III. SYMPTOMS AND DIAGNOSIS.—The disease is latent in many cases, but occasionally the patients complain of pain, varying in intensity from a dull feeling of pressure to a marked burning or pricking pain. The pain may be felt in the cervical region, between the scapulæ, beneath the sternum or epigastrium, or in the breast without any definite localization. The inflammation cannot be localized from the site of the pain. The pains sometimes arise spontaneously, or on making various movements of the spine (so that the patients often hold the head stiff and immovable), or on percussion of the spinous processes, or touching the lateral portion of the neck. Deglutition generally produces pain (odynophagia), which is not infrequently especially severe when the bolus passes a certain locality. Violent pain may also be produced by the introduction of a sound, and, under such circumstances, it may be diffuse or circumscribed.

Difficulty in deglutition is sometimes the direct result of the pain, but it may also be produced by reflex spasm of the muscular coat of the œsophagus. The patients experience the terrifying sensation as if the food remains sticking in one spot; they endeavor ineffectually to force it down; their terror increases; not infrequently reflex spasms of the muscles of the respiration occur, and even general convulsions. After vain attempts at deglutition, the bolus is regurgitated, occasionally coated with a muco-purulent or bloody mass. The sensation of the presence of a foreign body is sometimes experienced.

In examination with the sound, numerous epithelium cells of the mucous membrane often adhere to the fenestra of the instrument. The examination is painful, and often causes reflex spasm, so that the sound is held fast until the spasm relaxes. If the examination is performed carefully, the presence of streaks of blood upon the sound would favor the diagnosis of ulceration of the mucous membrane. Examination with the sound should not be employed, however, except in cases of urgent necessity.

The epithelium is sometimes desquamated to such an extent that it is expelled in the shape of a tube. Recovery occurred in a case of this kind reported by Birch-Hirschfeld.

Slight fever is sometimes present. The feeling of thirst is often greater than would be expected from the intensity of the fever.

IV. TREATMENT.—We should first attempt to remove the causes of the disease, and then directly combat the inflammation. If the pain is violent, an injection of morphine should be made into the side of the neck, or under the integument of the back near the spinal column, or narcotics may be given internally. The inflammation may be combated by the ingestion of pieces of ice, or small swallows of milk and ice. Not much can be expected from external derivatives (leeches, cups, blisters, mustard poultices, etc.). Solid food should be prohibited, and, if necessary, nutritive enemata employed.

In chronic catarrh, we may resort, if the patient is free from pain, to the introduction of a sound smeared with an astringent salve (R Acid. tannic., gr. xv.; vaselin., ℥ iij.; or argent. nitric., gr. viiiss.; vaselin., ℥ iij.). The operation should be performed after supper, and in the morning before breakfast.

4. *Phlegmonous Inflammation of the Œsophagus.*

This very rare disease leads to the formation of pus in the sub-mucous connective tissue of the Œsophagus. In the beginning, this tissue appears infiltrated with pus, and accordingly thickened; later it contains circumscribed accumulations of pus. The abscess often protrudes into the lumen of the Œsophagus, and causes stenosis. The epithelium over the abscess may be partly desquamated, and the mucous membrane is often congested. The muscular coat generally presents a normal appearance, though the microscope often reveals slight infiltration with pus. Perforation of pus occurs towards the inside alone; it not infrequently ruptures in several places at the same time. If recovery occurs, these openings generally cicatrize, but in some cases they remain open, the cavity of the abscess becomes lined with epithelium, and thus a sort of intraparietal diverticulum is produced. This may persist for a long time without causing any special disturbance.

The inflammation has been known to extend from the submucous layer of the Œsophagus to that of the stomach and vice versa.

The following are the causes of the disease: impacted foreign bodies, acute Œsophagitis produced by poisons, perhaps diphtheria of the canal, variola, phthisis, phlegmonous gastritis. The most frequent cause is suppuration in the vicinity of the canal, inasmuch as it extends to the submucous layer and there produces secondary suppuration. This category includes: tuberculosis of the spine, abscess of the lymphatic glands, and suppuration of the cartilages of the larynx. In some cases, the abscess perforates not only into the Œsophagus, but also into other cavi-

ties, for example, the air passages, thus producing an abnormal communication between the œsophagus and air passages. In some cases, no cause can be ascertained.

The disease can hardly be recognized during life.

5. *Corrosive Inflammation of the Œsophagus.*

Corrosive œsophagitis is that form of inflammation which is produced by toxic irritants—usually by mineral acids and alkalies.

The degree of inflammation depends on the nature of the poisonous substance and its degree of dilution. The changes sometimes involve the entire circumference of the organ, sometimes they appear in a band which runs along its entire anterior surface.

In cases of relatively brief action of the poison, the changes may be confined to the upper layers of epithelium. These are found corrugated, desquamated, whitish, desiccated, and present a certain resemblance to a croupous membrane.

In poisoning with alkalies we usually find a peculiar smeary, soap-like mass, produced by marked swelling of the epithelium.

If the irritation is more marked, the inflammation extends to the submucous connective tissue and phlegmonous œsophagitis is produced. If the irritant action is very intense, necrosis of all the layers of the œsophagus is produced, so that we find brittle, pulpy, reddish-brown or blackish shreds of tissue. The inflammation and necrosis extend not infrequently to the parts around the œsophagus.

When the changes affect the epithelium alone, *restitutio ad integrum* is the usual result, unless death is threatened by lesions of other organs. If the submucous layer is affected to a considerable degree, stenosis may be expected after the occurrence of cicatrization. Death generally takes place rapidly if extensive necrosis has been produced. After poisoning with tartar emetic the changes consist of simple inflammation; in other cases, of ulceration or pustulation of the œsophageal mucous membrane.

The symptoms of corrosive œsophagitis consist of pain, difficulty in deglutition, increased thirst, cough, and strangling. The latter act expels desquamated and mortified shreds of the wall of the organ. In some cases, almost the whole of the mucous membrane (even together with a portion of the gastric mucous membrane) has been desquamated in continuity. Cauterization and necrosis within the mouth and pharynx and upon the lips are noticeable, and sometimes excoriations run from the angles of the mouth across the cheeks, evidently because the poison ran out of the mouth. It is a notable fact that, in some cases, pain is experienced only at the outset, and later may disappear almost entirely.

The diagnosis follows from the clinical history and the appearances of the buccal cavity. If the history cannot be obtained, the application of blue and red litmus paper to the buccal mucous membrane will decide whether the poison is an acid or alkali.

The prognosis depends on the degree of destruction, but it should always be made with caution, since the development of stenosis does not occur, as a rule, until the lapse of many weeks.

The inflammation should be treated by giving small pieces of ice, milk and ice, and by external applications of ice. Morphine may be given internally or subcutaneously to relieve the pains. In poisoning with acids, we may give magnesia (gr. lxxv. to a glass of milk and ice, one

tablespoonful every ten minutes), in order to neutralize the poison, and diluted vinegar in ice-water in poisoning with alkalies. The patient should be nourished by means of rectal injections.

6. *Round (Peptic) Ulcer of the Œsophagus.*

Ulcers which are entirely similar to round gastric ulcers are sometimes found in the lower part of the Œsophagus. These will be produced if active gastric juice enters the Œsophagus during the act of vomiting, and remains there a sufficient length of time to exercise a digestive and destructive effect. The symptoms are also similar to those of the round gastric ulcer, viz., hæmatemesis, bloody passages, perforation of the wall of the Œsophagus, formation of cicatrices and stricture. The treatment is the same as that of the round gastric ulcer.

7. *Cancer of the Œsophagus.*

I. ANATOMICAL CHANGES.—Cancer of the Œsophagus is almost always primary; secondary cancer is rare, and generally extends from the stomach, more rarely from the pharynx. The primary cancer is situated occasionally in the mediastinum.

Primary cancer of the Œsophagus is almost always a flat pavement-epithelium cancer, sometimes hard, sometimes juicy. The occurrence of scirrhus or encephaloid cancer in this locality is doubtful.

It is situated generally in the lower third of the canal, then in the middle, and least frequently in the upper third. The sites of predilection are the portion immediately above the cardiac end of the stomach, the point where the canal crosses the left bronchus and the region behind the cricoid cartilage. All these places are particularly subject to irritation during deglutition.

The cancer may appear in patches (insular), or in the shape of a girde (circular). The former constitutes the beginning of the disease; the latter form produces stenosis in two ways: by preventing dilatation of the Œsophageal wall, and by proliferation into the lumen of the canal.

As a rule, there is but one growth, which varies from three to ten cm. in height. In rare cases the entire canal is involved in the cancerous process.

In one case, Zenker found that the middle of the organ, over an extent of four cm., was alone free from the cancerous degeneration.

Carmalt states that not alone the epithelial cells of the mucous membrane, but also those of the excretory ducts of the mucous glands proliferate into cancer cells. The muscular coat adjacent to the cancer is generally hypertrophic; later it becomes infiltrated with masses of the neoplasm.

The Œsophagus is sometimes dilated above the tumor, but this dilatation is not so frequent as is generally believed. The tracheo-bronchial and mediastinal lymphatic glands are usually enlarged and cancerous. The deeper cervical glands are more rarely affected, the peripheral cervical glands usually escape.

If necrosis of the cancer occurs, a previous stenosis may disappear. As a rule, however, the improvement is only temporary. The necrosis is sometimes so extreme that careful observation is necessary to discern

the remains of the cancer, and Rokitsansky even believed that recovery was possible from the formation of a cicatrix.

In some cases, the ulcerative degeneration spreads to adjacent organs, and may lead to abnormal communication with the air passages, lungs, pleura, pericardium, cavities of the heart or the large vessels.

Perforation occurred twenty-seven times among forty-four cases collected by Petri. The following table of fifty-three cases of perforation is furnished by Schneider :

Perforation into the trachea,	21 times.
“ “ “ lungs,	16 “
“ “ “ bronchi,	16 “

The neoplasm may be propagated in various ways. It often spreads directly to adjacent organs, viz., the stomach, pharynx, pleura, lungs, bronchi, trachea, mediastinal cellular tissue, pericardium, myocardium, aorta, and vertebræ. The vertebræ are sometimes perforated and the spinal cord may be compressed. The cancer may also extend from the mediastinum into the spinal canal through the intervertebral foramina.

In other cases, the growth spreads by dissemination, probably through the agency of the lymphatic channels. In this way, the tumor extends to the mucous membrane of the œsophagus itself, the pleura, pericardium, and peritoneum.

True metastasis takes place occasionally, and secondary nodules are observed in the lungs, liver, kidneys, suprarenal capsules, pancreas, medulla of the bones, and lymphatic glands.

II. ETIOLOGY.—Primary cancer of the œsophagus is a disease chiefly of old age, being most frequent from the age of forty to sixty years. In the majority of cases, it occurs in the male sex.

The influence of heredity is questionable. Burning during deglutition, injury from foreign bodies, abuse of alcohol, and chronic diseases of the stomach, are mentioned as exciting causes. C. Neumann reported a case in which the cancer followed an ulcerative and cicatricial process.

III. SYMPTOMS.—As a rule, the symptoms develop slowly and gradually, and our attention may first be attracted by the signs of œsophageal stenosis. At the same time, we are often struck by the emaciation and cachectic appearance of the patient.

The disease sometimes begins with very violent pains, which are partly spontaneous, partly the result of the act of deglutition. I recently treated a man who suffered from the most violent pains for nearly a year before the signs of stenosis were demonstrated. The patient located the pain in the lower part of the œsophagus, although examination with the sound and the autopsy showed that the cancer was situated in the upper third of the canal. In some cases the pains occur only at night; they radiate occasionally into the limbs and along some of the intercostal spaces.

Phonation is interfered with in not a few cases, on account of paralysis of the recurrent laryngeal nerves; the act of coughing and straining also suffers under such conditions. The paralysis of the vocal cords is more often unilateral, but can hardly be recognized, except with the aid of the laryngoscope. Sometimes the tumor compresses the nerves, sometimes it proliferates into them. The paralysis may be a very early symptom and even precede the signs of stenosis.

If the cancer is situated in the cervical part of the œsophagus, it may be visible as a painful, infiltrated swelling; when it extends to the pharynx it may be visible through the buccal cavity. Examination with

the sound should be resorted to if the cancer is situated more deeply. In this manner we first determine the situation and degree of an œsophageal stenosis. The particles which remain adherent to the fenestra of the sound should be carefully examined, since they can be recognized very often as fragments of cancer, on account of the so-called pearly globules (Fig. 7) which they contain. The regurgitated masses also contain particles of cancer, in addition to food, pus, and blood. The presence of numerous epithelial cells with two nuclei is also suspicious, even if the pearly globules are absent. The sound should be introduced carefully, in order to avoid perforation and hemorrhage. Auscultation furnishes signs similar to those obtained in œsophageal stenosis (vide page 31).

Fig. 7.



Pearly globule from cancer of the œsophagus, which had remained in the fenestra of the sound.
Magnified 275 diameters.

The majority of patients complain of great thirst. If the œsophagus is markedly stenosed, the food regurgitates, the patient's hunger is never satisfied, and he grows weaker and weaker. The digestive power of the stomach is very much diminished, and the food may remain undigested for many hours. Riegel showed that the carcinoma destroys the hydrochloric acid of the gastric juice, and thus makes the latter inefficient. The patients generally complain of obstinate constipation. An increased amount of indican is often found in the urine (if a test tube is filled with equal amounts of urine and hydrochloric acid, and one to three drops of a fresh solution of chloride of lime are added, the urine will assume a reddish or bluish color). The amount of urea is diminished. Marchand states that asthma-like or stenocardic attacks are occasionally observed. Pulmonary phthisis develops not infrequently. Marantic œdema and venous thrombosis develop not infrequently with the increasing loss of power.

The disease may last many months. According to Lebert, the average duration is thirteen months.

The fatal termination results from starvation, associated with the signs of increasing Œsophageal stenosis (these symptoms may improve temporarily if portions of the growth are cast off on account of ulceration), from increasing marasmus, a fatal hemorrhage, Œsophageal perforation, or complicating pneumonia, pulmonary gangrene, pleurisy, and pericarditis, or severe spinal paralysis. Some patients present symptoms of pyæmia.

IV. DIAGNOSIS.—The diagnosis consists of the demonstration of a stenosis and the recognition of its cancerous character. Concerning the former problem, we refer to the remarks on Œsophageal stenosis (Vol. II., page 31). The carcinomatous character of the stenosis is recognized from the previous history, the associated symptoms, general cachexia, age, and the presence of fragments of cancer in the vomited masses or upon the sound.

V. PROGNOSIS.—This is unfavorable, as in all other forms of cancer.

VI. TREATMENT.—Special attention should be paid to a suitable diet (vide Vol. II., page 32). The pain may be relieved by narcotics. Careful sounding not infrequently produces marked diminution of the disturbances of deglutition for a considerable period. Ziemssen advises the daily use of the sound.

Surgical text-books must be referred to for detailed information concerning operative procedures.

Resection of the Œsophagus may only be performed if the cancer is situated in the cervical portion of the Œsophagus. This is also true of Œsophagotomy. Early gastrotomy seems to be more advisable, even if it is intended to follow this operation by resection of the Œsophagus.

8. Hemorrhages from the Œsophagus.

Hemorrhages may take place either into the tissue of the Œsophagus itself, and then possess merely an anatomical interest, or upon the free surface of the mucous membrane, in which event they may be recognized during life, if circumstances are specially favorable.

The hemorrhage is very rarely the result of external injury, much more frequently of injury internally by ingested foreign bodies. As a rule, it does not take place immediately after the ingestion of the foreign body, but ulceration of the mucous membrane first occurs, and this constitutes the immediate cause of the hemorrhage. If it takes place from vessels of the Œsophagus itself, it is less serious than if the large vessels near the organ are eroded (aorta, pulmonary artery, carotid, subclavian, intercostal arteries, vena cava, hemiazygos, and even the cavities of the heart). Unskilful use of the sound may also produce traumatic hemorrhage, and this may prove serious if the patient is suffering from a vascular cancer of the Œsophagus or from aortic aneurism. Closely allied to traumatic hemorrhages are those produced by irritant poisons. Ulcerative processes of the Œsophagus may also give rise to hemorrhage, either from the vessels of the Œsophagus itself or adjacent large vessels which have undergone perforation. Spontaneous rupture and softening of the Œsophagus also give rise to hemorrhage. In some cases it is the result of varicose dilatation of the Œsophageal veins, either from rupture of the veins or from the extension of an ulcerative process of the mucous membrane to the subjacent veins. This condition is observed most fre-

quently in cirrhosis of the liver, because the coronary veins of the stomach, and the veins of the lower part of the œsophagus attempt to convey a part of the blood of the portal vein to the vena azygos, independently of the circulation in the liver. Klebs noticed varicose veins in the submucosa of the œsophagus in syphilitic hepatitis, and Zenker observed a similar condition in tight-laced fissure of the liver and marked senile atrophy. Old age, in itself, seems to predispose to varicose dilatation of the œsophageal veins. In some cases the cause of the development of the varices cannot be recognized. (Esophageal hemorrhages sometimes originate in surrounding parts, for example, in aneurisms of the aorta.

The symptoms of œsophageal hemorrhage consist of hæmatemesis and evacuation of black or bloody masses in the stool. The latter occur only when the hemorrhage is very profuse. Rapid death sometimes follows with signs of internal hemorrhage (pallor and coolness of the skin, imperceptible pulse, nausea, failure of the senses). Sometimes the first hemorrhage proves fatal, at other times the hemorrhage ceases, and death occurs in a subsequent attack.

The diagnosis can be made only when gastric and intestinal hemorrhage can be excluded.

The prognosis depends on the primary cause and the abundance of the hemorrhage.

The treatment consists of the ingestion of small pieces of ice, and the application of an ice-bag to the præcordial region and to the left of the spinal column; several subcutaneous injections daily of Ergotinum Bombelon (one-half syringeful diluted with an equal amount of water) should be made to the left of the spine, and five to ten drops of liq. ferri sesquichlorat. in a tablespoonful of water should be given every two hours. Symptoms of collapse should be combated by subcutaneous injections of camphor (camphoræ, gr. xv.; olei amygdalar., 3 iij. M. D. S. One syringeful t. i. d.), and the administration of champagne and ice.

For days no food except iced milk should be administered by the mouth, and the strength should be maintained by nutritive enemata.

9. *Perforation of the Œsophagus.*

1. Perforation of the walls of the œsophagus may occur from within outwards (primary) or vice versa (secondary). This process is more frequent in males, and at an advanced age. The thoracic portion of the œsophagus is most frequently affected, especially the part which is adjacent to the bifurcation of the trachea. This is owing, in part, to the fact that diseases of the bronchial and tracheal lymphatic glands play a prominent part in the etiology of the disease.

Among the causes of primary perforation of the œsophagus we must first mention traumatism. Thus it occurs not very rarely in examination with the sound, which penetrates the wall of the œsophagus, and enters a false passage (mediastinum, pleural sac, pulmonary cavity, etc.). Eras reported a case occurring in a mountebank who was able to pass three swords (fifty to fifty-five cm. in length) into the œsophagus. On one occasion the trick failed, and the point of one sword entered the mediastinum. Foreign bodies may lead to perforation either directly or indirectly. In the latter event perforation is preceded by ulceration. In some cases perforation depends upon toxic influences, or upon various forms of ulcerative processes (catarrh, diphtheria, tuberculosis, cancer, syphilis, etc.). (Œsophagomalacia will be discussed later.

Secondary perforation of the œsophagus is produced most frequently by cheesy and softened bronchial and tracheal lymphatic glands. In other cases it follows cold abscesses resulting from tuberculosis of the spine, or retropharyngeal abscess, phthisical cavities, pulmonary gangrene, or cancer. It is produced occasionally by diseases of the trachea, as in Steffen's case in which a tracheotomy canula, which had been left in too long, destroyed the posterior wall of the trachea, and finally perforated the œsophagus. Zenker observed perforation of the œsophagus by prominent tracheal cartilages as the result of excessive pressure of the trachea backwards by a goitre. Zenker also observed perforation by a gangrenous cyst of the thyroid gland. Finally, aortic aneurisms sometimes perforate into the œsophagus.

2. As a rule, there is but one point of perforation; more rarely there are several adjacent ones. The opening is sometimes round, sometimes slit-shaped, and in the latter event may be several centimetres in length. In some cases the perforation occurs gradually and spontaneously, in others it is produced slowly, sometimes by the act of vomiting.

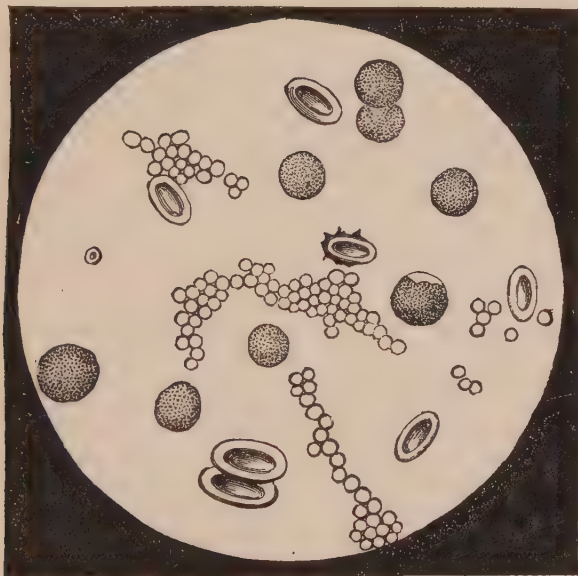
3. As a matter of course, the perforation causes an abnormal communication between the lumen of the œsophagus and adjacent organs. Hence the symptomatology is extremely variable. If the perforation takes place into the connective tissue of the mediastinum and neck, inflammatory and gangrenous processes are the inevitable result. The pus may then rupture externally, giving rise to an external œsophageal fistula, through which pass pus, ichorous fluid, and particles of food. In perforation into the large vessels, fatal hemorrhage occurs, attended usually with hematemesis. If the œsophagus opens into the air passages, deglutition is generally followed, in a little while, by violent cough and particles of food are expectorated. In one case, Œbernier saw the communication by the aid of the laryngoscope. Water mixed with powdered charcoal was given the patient to drink, and the particles of charcoal could then be seen in the trachea. Such communications are usually followed by pneumonic or gangrenous changes in the lungs, the result of aspiration of particles of food. If a communication has been formed between the œsophagus and a pulmonary cavity, a sound, whose introduction formerly soon met with an obstruction, apparently passes farther into the œsophagus, because it passes through the opening into the cavity. In addition, the ingestion of fluid may produce increased thoracic dulness by filling the cavity, and this again changes to a tympanitic percussion note when the fluid is expectorated. If a colored fluid has been ingested, the sputum will present a corresponding color. In addition, ichorous processes will soon develop in a cavity which previously had not produced a putrid secretion. If the cavity is gangrenous, its secretion may pass into the œsophagus, so that expectoration is replaced by putrid evacuations. If a communication forms between a previously healthy lung and the œsophagus, gangrenous changes develop in the lung. In one case I found, for a week, very numerous, colorless, double-contoured bacteria in the sputum, and then the sputum became gangrenous (vide Fig. 8). The patient suffered from œsophageal cancer which had perforated into the right lung. In one case Lesser found numerous cells of ciliated epithelium in the sputum, after the perforation into the lung had occurred. Communication with the pleural or pericardial cavities is shown by the development of pneumothorax and pyopneumothorax, or pneumopericardium and pyopneumopericardium, but these lesions may also precede the perforation. Cutaneous

emphysema rarely follows perforation of the œsophagus, because the perforation is generally preceded by inflammation in the mediastinum which prevents the entrance of air.

The beginning and end of the disease may be almost coincident, especially if a large vessel has been opened. In other cases the patients complain, at the time of perforation, of pain, anxiety, a sensation as if something had burst internally, and collapse. The perforation sometimes occurs so slowly that it may be overlooked, and according to Vigla, it may persist for several months.

4. The prognosis depends partly on the primary disease, partly on the mode of perforation. Recovery is possible.

FIG. 8.



Fungus spores from the sputum in a case of œsophageal cancer which had penetrated into the lung. Magnified 600 diameters.

5. The strength must be maintained by nutritive enemata, in order to allow cicatrization to occur.

10. *Spontaneous Rupture of the Œsophagus.*

1. Spontaneous rupture of the œsophagus, *i. e.*, that form which is not preceded by injury or disease, is extremely rare. Only fourteen cases have been reported.

The majority of the cases occurred in men, many of whom were addicted to excesses, particularly in the use of alcohol. Two cases also presented symptoms of pyæmia.

The rupture was often preceded by vomiting; in one case while the patient was ironing, in another during defecation.

2. The accident always occurred suddenly. The patients cried out that something had burst internally, and complained of intolerable pain in the lower part of the spine and between the shoulders; in addition,

fear of impending death, pale face, cool extremities, and imperceptible pulse. Nausea and vomiting were frequently observed at the onset, occasionally hæmatemesis. Cutaneous emphysema soon occurred, at first around the neck, then over larger areas. Death generally occurred in collapse within twenty-four hours; in one case life was maintained for a week.

3. The rupture was generally longitudinal, beginning not far from the cardiac end of the stomach, more than five cm. in length, affected all the coats of the organ, and usually led first into the mediastinum and then into one or both pleural cavities. The latter contained the contents of the stomach. In one case two longitudinal perforations were present, in another the opening was circular.

Zenker showed that enormous force is necessary to rupture the œsophagus, and he believes that œsophagomalacia was present in the cases under consideration.

4. The diagnosis is always difficult. It may possibly be made if there is a sudden development of hæmatemesis, pleural effusion, pneumothorax, and cutaneous emphysema. At the same time we must be able to exclude previous disease of the œsophagus, perforation of the stomach, and other causes of internal hemorrhage.

5. The prognosis is bad. If the pains are very violent, treatment must be confined to the use of narcotics and to the administration of stimulants to relieve the collapse. All remedies should be administered subcutaneously.

11. *Softening of the Œsophagus. Œsophagomalacia.*

1. Softening of the œsophagus almost always affects the lower part of the canal. It is generally associated with gastromalacia. If the mucous membrane is alone affected, it is converted into a soft gelatinous mass of a grayish, yellowish, brownish, or blackish-green color.

If all the coats are affected, there is danger of rupture. This takes place most frequently into the left pleural cavity; even the lung may be implicated. Softening processes and ecchymoses are sometimes found upon the aorta and in the posterior mediastinum.

In the majority of cases the lesion is produced post-mortem as the result of digestion by the gastric juice. But some cases are of vital origin, or at least have developed during the agony. This is especially true of those cases known as brown softening, and the infarctions, which are often observed, also indicate a vital origin. For, if the contents of the stomach, especially the gastric juice, regurgitate into the œsophagus, self-digestion will readily take place as soon as the circulation is partly interrupted. The gastric juice then unfolds its digestive action because it is not neutralized by the alkaline blood.

2. The diagnosis during life is always difficult, if not impossible, especially since such patients may not be moved much in order to avoid rupture of the œsophagus. Under the most favorable circumstances the condition may be recognized by hæmatemesis and sudden signs of pneumothorax or hydropneumothorax.

3. Œsophagomalacia is especially apt to develop in basilar meningitis and chronic cerebral diseases in general; also whenever the death struggle is prolonged.

12. *Sprue in the Œsophagus. Œsophagomycosis oidica.*

1. *Oidium albicans*, when it appears upon the œsophageal mucous membrane, is almost always propagated from the buccal cavity and pharynx. It is observed in feeble individuals suffering from acute or chronic diseases (cholera infantum, phthisis, diabetes, typhoid fever, pyæmia, etc.).

2. It sometimes appears in the form of more or less large, irregular patches, as long stripes, or as complete casts of the œsophagus. It may also proliferate to such an extent as to fill the canal with a solid plug. According to Bompard, the fungus develops particularly in the upper and lower parts of the canal.

It rarely extends into the œsophagus, since cylindrical and ciliated epithelium seem to resist its proliferation. Grawitz attributes the immunity of the stomach to the acid reaction of its mucous membrane. Sprue sometimes extends into the larynx and deeper air passages.

The masses of fungus appear as white, yellow, or yellowish-gray deposits upon the mucous membrane, and may be removed. The subjacent mucous membrane is occasionally congested, and may even present superficial ulceration.

Wagner noticed that the fungus penetrated between the uppermost layers of epithelium, and then proliferated freely in the succulent middle layers, where it gave rise to atrophy, so that sometimes only the nuclei of the cells remained. Wagner also noticed that it entered the mucosa, and even the blood-vessels. Zenker found the fungi in the vessels of the brain, whither they had probably been carried as emboli, and had then continued to proliferate.

3. In many cases sprue of the œsophagus presents no symptoms. In others there are disturbances of deglutition which occasionally end in symptoms of complete occlusion. Emboli in the brain may give rise to grave cerebral symptoms.

4. The diagnosis can only be made when fungi are found in the vomited matters, and it is certain that they do not come from the mouth or pharynx. In examination with the sound, masses of the fungus may remain in the fenestra.

5. The prognosis is often unfavorable on account of the primary disease, but the fungus itself may give rise to severe symptoms (dysphagia, aphagia, cerebral symptoms). Slight proliferations possess no significance.

6. Treatment consists of the prophylactic measures against the development of sprue in the mouth (vide Vol. II., page 13). If there is difficulty of deglutition, the fungus should be removed mechanically by the administration of emetics.

13. *Paralysis of the Œsophagus.*

1. Little is known concerning this affection. Its causes include diseases of the brain and cervical cord (hemorrhage, tumors, multiple sclerosis, bulbar paralysis, ataxia, progressive general paralysis). A few cases have been produced by compression of both pneumogastric nerves by swollen lymphatic glands. Diphtheria, syphilis, lead poisoning, alcoholism, cold, ingestion of hot food, and mental excitement have also been mentioned as etiological factors. Hysteria is a rare cause.

2. The symptoms consist of difficulty of deglutition. In addition, we

sometimes notice a feeling of oppression, palpitation, dyspnœa, partly the result of excitement, partly the result of pressure of the œsophagus (which is distended by the food) upon the heart, lungs, and recurrent laryngeal nerves. Large, firm boluses are often swallowed more readily than small, fluid ones. Some patients swallow better in the upright position, others help the bolus along with the aid of a stick. Regurgitation of food sometimes occurs or the latter enters the stomach with a rumbling noise. In examination with the sound, the tip of the instrument is found to be freely movable on all sides. Auscultation shows that deglutition is slowed.

3. The prognosis depends upon the cause.

4. We must secure suitable nourishment for the patient and, if necessary, use the œsophageal sound for this purpose. The faradic current sometimes produces good effects, but the current should not be too strong in order to avoid irritation of the pneumogastriæ. One pole is applied to the cervical spinal processes, the other within the œsophagus, either by means of a laryngeal electrode (vide vol. I., Fig. 50), or a similar but longer and more flexible one. The current should be applied shortly before dinner.

14. *Spasm of the Œsophagus. Œsophagismus.*

1. Spasm of the œsophagus occurs in many central neuroses (hysteria, hypochondria, epilepsy, chorea, tetanus), and is an important symptom of hydrophobia. It is observed occasionally in very nervous individuals who, having been bitten by a dog, live in dread of the onset of hydrophobia. Mental excitement (fright, terror, joy) is also mentioned among the causes. In many cases it has a reflex origin. Sudden spasm occurs not infrequently, even in robust individuals, during examination with the œsophageal sound. Œsophagismus may be produced by imperfectly masticated food, foreign bodies or very hot food; also by inflammation and cancer of the organ. Pharyngeal polypi, pharyngitis granulosa, and chronic angina may also give rise to the disease. It also occurs in diseases of the stomach and intestines, and is observed particularly in women who are suffering from uterine diseases. It occurs occasionally during pregnancy and lactation. In some cases the disease has been attributed to injury of the breast; also to poisoning with belladonna and stramonium. Romberg observed it during arthritis.

2. The disease is characterized by a disturbance of deglutition, associated with a feeling of constriction and occasionally of pain. If the spasm affects the upper part of the œsophagus, the food is regurgitated at once. In addition there may be a feeling of oppression, palpitation, dyspnœa, sometimes loss of consciousness, and general convulsions. The spasm sometimes occurs at the mere thought of eating. The affection may last for days, weeks, or even months.

In examination with the sound a resistance is encountered which often ceases suddenly. On auscultation, the sound produced by deglutition can only be followed as far as the site of spasm, but the latter often changes.

The diagnosis is usually easy. The prognosis is almost always favorable, except in hydrophobia, or when the disease is the result of anatomical lesions of the central nervous system.

Therapeutic measures should be directed mainly against the primary disease. In addition, we may attempt to relieve the spasm by the sub-

cutaneous administration of narcotics. The application of the constant current is often indicated (cathode to the cervical spine, anode within the œsophagus, a feeble current one to two minutes daily). Recovery is sometimes effected by careful repeated introduction of the sound.

PART III.

DISEASES OF THE STOMACH.

A. GASTRIC AFFECTIONS ASSOCIATED WITH ANATOMICAL LESIONS.

1. *Hemorrhage of the Stomach.*

I. ETIOLOGY.—Hemorrhage is probably much more frequent than appears, since, as a rule, a diagnosis can only be made if the hemorrhage is not too slight in extent. The following are the causes of the disease :

a. Injuries, chemical and thermal irritants.

This category includes a blow or fall upon the gastric region, ingestion of sharp substances or of hot food, acids, alkalies, or other irritating substances.

b. Diseases of the vessels of the stomach.

These include varicose or aneurismal dilatation of the vessels of the mucous membrane, and embolic processes.

c. Ulcerative affections of the walls of the organ, particularly hemorrhagic erosions, round gastric ulcers, cancer, phlegmonous gastritis, and also tubercular ulcerations.

d. Excessive arterial fluxion of the mucous membrane.

Hence hemorrhage occurs not infrequently in violent gastritis. This category also includes the vicarious gastric hemorrhage which takes the place of menstruation, and is also said to occur at times instead of an habitual flux.

e. Stasis in the portal vein which impedes the flow of blood from the gastric veins, or local stasis in the mucous membrane of the stomach.

This is sometimes the result of occlusion of the trunk of the portal vein (pylophlebitis) or of some of the branches within the liver, as the result of cirrhosis, tumors, pigment emboli in protracted intermittent fever, dilatation of biliary capillaries following stasis of bile, sometimes the result of circulatory disturbances following chronic diseases of the respiratory or circulatory apparatus. The causes of local stasis include protracted or violent vomiting or straining (childbirth). Gastric hemorrhage is also said to occur occasionally in pregnant women as the result of stasis.

f. Nervous influences (vaso-motor?).

It has been shown that injury of the central nervous system may produce hemorrhage of the stomach in animals. This has also been observed during the course of hysteria and general paresis of the insane.

g. Aneurisms and abscesses which rupture into the stomach, or perforation of a gastric ulcer or cancer into the cardiac cavities or the large vessels.

Thus gastric hemorrhage has been observed from rupture of aneurisms of the aorta or celiac axis, perforation of a cold abscess following vertebral caries, perforation of a round gastric ulcer into the left heart.

h. Infectious diseases.

Gastric hemorrhage occurs occasionally in yellow fever, small-pox, measles, scarlatina, etc., when the signs of so-called dissolution of the blood make their appearance. It has been observed occasionally in intermittent fever. Kron reported a case in which it appeared every third day, and ceased after the administration of quinine. Sometimes it does not appear until the period of malarial cachexia. It is associated generally with grave changes in the blood, resulting in abnormal permeability of the walls of the vessels and excessive diapedesis of red blood-globules.

i. Blood diseases.

Gastric hemorrhage is observed in hæmophilia, morbus maculosus Werlhofii and scurvy, and also in progressive pernicious anæmia. The connection between the hemorrhage and the primary disease is probably the same as in the diseases mentioned under *h*.

k. Poisons.

This category includes poisoning with acids, alkalies, phosphorus, arsenic, etc. and also uræmia and cholæmia.

Gastric hemorrhage is most frequent from the fifteenth to the fortieth years of life. It is extremely rare in childhood. A special form of the disease, known as *melæna neonatorum*, occurs in the new-born, and will be discussed later.

The disease is more frequent in women than in men, because the round ulcer, which is the chief cause of gastric hemorrhage, is especially frequent in the female sex. Moreover, menstruation is not without influence on the development of gastric hemorrhage.

The hemorrhage sometimes appears to be spontaneous, sometimes it is preceded by bodily or mental excitement, overloading of the stomach, ingestion of indigestible food, etc.

II. ANATOMICAL CHANGES.—The anatomical changes, so far as regards the hemorrhage, depend upon the abundance of the latter. If it is very profuse, the stomach is found tense, and the contents shine through with a bluish or blackish color. Upon opening the organ we find blackish clots, which sometimes present the shape of the stomach. If the blood is removed, and the mucous membrane washed with water, the surface is found to be very pale. This is also true of the other viscera. When the hemorrhage has not been the immediate cause of death, fatty degeneration of the heart-muscle, kidneys, and other glands can often be demonstrated.

The source of the hemorrhage cannot always be discovered, particularly in capillary hemorrhages. In other cases, an eroded vessel is found, sometimes partly occluded by a thrombus. The site of the hemorrhage can sometimes be ascertained by the injection of a colored fluid into a main artery.

If the hemorrhage is small in extent and has developed gradually, the contents of the stomach are chocolate-colored, like coffee grounds, inky and sooty, exactly like the vomited matters.

The lesions which give rise to the hemorrhage cannot be discussed here.

If the hemorrhage has been profuse, bloody, blackish or tarry contents are often found in the intestines, sometimes in the upper air-passages, into which they have passed during the act of vomiting.

III. SYMPTOMS.—Slight hemorrhages often occur without any symp-

toms. The blood-globules are then entirely dissolved by the gastric juice, and leave no recognizable traces.

But if the hemorrhage is larger in amount, this change can no longer be effected by the gastric juice, and the evacuations from the bowels assume a bloody character. The patients usually discharge tarry, blackish, sometimes soft, sometimes very hard masses, which often have a pestilential odor.

The stools should therefore be examined in all conditions which may possibly give rise to gastric hemorrhage, particularly when sudden pallor of the skin, small pulse, syncopal attacks, etc., arouse the suspicion of hemorrhage. The evacuations are sometimes accompanied by colicky pains and tenesmus.

In many cases gastric hemorrhage is attended by hæmatemesis. If the hemorrhage is small in amount, the vomited masses consist of mucus, or chiefly of food, mingled with dots and streaks of fresh blood. If the hemorrhage is more profuse, but the blood has remained for some time in the stomach, the coloring matter of the blood is converted into hæmatin by the hydrochloric acid of the gastric juice, and the vomited masses have a brownish, chocolate, or inky color. This is most frequent in cancer of the stomach, but is also observed in gastric ulcer, cholæmia, poisoning, etc. Under the microscope we find discolored, dentate or degenerated red blood-globules, mixed with food (vide Fig. 9).

If the hemorrhage is very profuse, brownish-black, non-aërated clots, usually of an acid reaction, are vomited together with articles of food. Several pounds may be vomited. If a large artery has been opened, the blood occasionally has a bright, arterial appearance, and the red blood-globules are usually found unchanged.

Gastric hemorrhage may occur without hæmatemesis, and the latter is sometimes observed in cases in which the blood does not really originate in the stomach, for example, in profuse intestinal hemorrhage, or when blood flowing from the nose, pharynx, œsophagus, or air passages has been swallowed and then vomited.

Vomiting of blood and bloody evacuations are often associated with one another.

During the hemorrhage many patients experience a sensation as if something warm were flowing into the stomach. Not infrequently, there is a feeling of abnormal abdominal pulsations. The patients often complain of a feeling of constriction or fulness in the gastric region.

A feeling of warmth soon ascends along the œsophagus, a tendency to vomit follows, a sweetish taste in the mouth becomes noticeable, and the patient finally vomits blood.

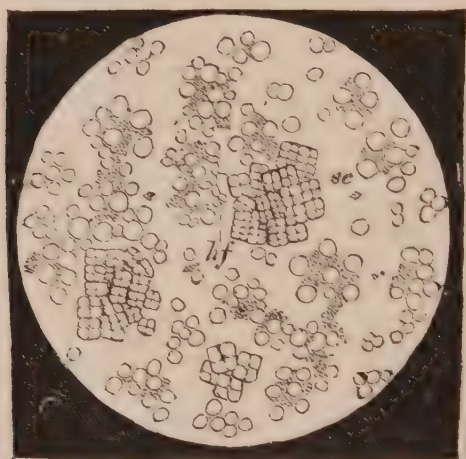
If the hæmatemesis is very violent, masses of blood sometimes burst from the mouth and nose. The blood not infrequently enters the air passages, and gives rise to cough or danger of suffocation. The accident always exerts a depressing influence upon the patient.

The symptoms usually begin or are accompanied or followed by signs of rapid loss of blood. The patients grow as pale as death. They complain of faintness, dizziness, ringing in the ears, spots before the eyes, a film over the eyes; the pulse grows small, occasionally imperceptible. If the hemorrhage is very considerable, death may occur, though not a drop of blood escapes by vomiting or an evacuation from the bowels.

There is almost always a tendency to relapse, so that many patients experience a number of attacks.

The sequelæ of a profuse gastric hemorrhage may last a long time. For weeks, the patients continue to be as pale as death. (Edematous swelling of the ankles, eyelids, and conjunctiva often develops within a few hours. There is marked apathy and somnolence, and at the same time inability to sleep at night. The pulse is not infrequently markedly dirotic. Systolic anæmic murmurs develop in the heart. In one case I observed a pericardial friction murmur, which I attempted to explain by subpericardial extravasations of blood. Venous murmurs are heard over the bulb of the internal jugular vein, and over the crural vein immediately below Poupart's ligament. A systolic arterial sound is heard on auscultation of the larger arteries. The blood obtained on pricking the tip of the finger has a serous color, and contains scanty red blood-globules, which are very pale and irregular in size and shape (poikilocytosis). A strikingly large number of elementary corpuscles are found

FIG. 9.



Coffee-grounds vomited matters in a woman æt. 32 years, suffering from cancer of the stomach. The red blood-globules have lost their color, and form colorless circles. *sc*, sarcina ventriculi; *hf*, a group of yeast cells. Enlarged 275 diameters.

occasionally in the blood. The white corpuscles are sometimes slightly increased in number (leucocythæmia).

Marked desquamation of the skin, loss of hair, and albuminuria are less frequent events. In a number of cases, I observed intolerable *fœtor ex ore*, marked dryness of the mouth, and unquenchable thirst. Amaurosis has been observed in a number of cases after profuse losses of blood, particularly after hæmatemesis. Its causes are unknown, but it has been attributed to central hemorrhages, œdema of the sheath of the optic nerve, serous infiltration, and circulatory disturbances in the retina. The latter are proven by the retinal hemorrhages observed in some cases. Neuroretinitis, and later, atrophy of the optic nerve, have also been seen. The visual disturbances develop a few days after the hemorrhage, and, after a while, may subside more or less completely. They are said to be present particularly in those cases in which pain in the occiput and stiffness of the neck developed after the hæmatemesis.

IV. DIAGNOSIS.—When the hemorrhage is so profuse that death ensues before the occurrence of hæmatemesis or bloody evacuations, a probable diagnosis can only be made when the signs of internal hemorrhage suddenly develop in a patient who had long presented gastric symptoms.

If bloody evacuations alone occur, the statements of the patient may only be accepted if he presents an anæmic appearance. Blackish stools, which are erroneously supposed to contain blood, are passed after the administration of ferruginous preparations and bismuth. In doubtful cases, water, which is poured over the stool, will assume a bloody color if the stool contains blood. Or we may employ the microscope or spectroscope. As a matter of course, the passage of a bloody stool is not proof of a gastric hemorrhage. Such a conclusion is only warranted when we can exclude intestinal hemorrhage and the passage of blood into the stomach. The presence or absence of gastric symptoms must be employed not infrequently in making a differential diagnosis.

Mistakes may also be made concerning hæmatemesis. I recently observed a questionable case of cancer of the stomach, in which an ominous sooty mass was vomited, and seemed to favor the diagnosis. On microscopical examination of the vomited matter, it was found to contain, not blood-globules, but crystals of bismuth. Iron preparations may also give rise to pseudo-hæmatemesis.

Blackish-red and blood-like vomiting may also occur after eating cherries, beets, sausage, claret, etc. A mistake is hardly possible if the physician sees the vomited matter. In doubtful cases, the previously mentioned tests will decide.

Malingers sometimes swallow blood, and then vomit it with the aid of emetics, but signs of acute anæmia are absent in such cases. The individual is apt to contradict himself or lays a trap for himself by exaggerating the symptoms in the attempt to appear sick.

A mistake is possible in the new-born, inasmuch as they may swallow blood, while nursing, from a wound of the nipple, or it may have entered the mouth from the genital passages of the mother during delivery.

For the differential diagnosis of hæmatemesis and hæmoptysis, vide Vol. I., page 256.

Hæmatemesis may be attributed to gastric hemorrhage only in those cases in which hemorrhages from the nose, pharynx, œsophagus, or intestines can be excluded.

The amount of the hemorrhage may enable us to judge whether it has come from a large or small vessel. The hemorrhage may be assumed to be arterial if the blood is bright red.

V. PROGNOSIS.—Death is rarely an immediate result of profuse hemorrhage from the stomach. It has also been observed a number of times in round gastric ulcer that the pains, feeling of fulness, anorexia, and nausea diminished after a gastric hemorrhage. Furthermore, hæmatemesis has been known to cause diminution of ascites resulting from cirrhosis of the liver. Nevertheless, gastric hemorrhage is not a favorable symptom. Its causes often possess a grave significance, and the symptom itself enfeebles the organism and accelerates the failure of the vital energies.

VI. TREATMENT.—After a gastric hemorrhage occurs, the patient should be placed in bed, relieved of all tight articles of clothing, and kept absolutely quiet. A subcutaneous injection of ergotinum Bombe-

lon (one-half syringeful with an equal amount of water) should be made in the region of the stomach, an ice-bag applied to the epigastrium, and small pieces of ice swallowed. If syncopal attacks make their appearance, the head should be held as low as possible, the face and chest sprinkled with cold water, ammonia or ether held to the nostrils, a mustard poultice applied to the calves, and camphor (gr. xv., ol. amygdalar. 3 iij., one syringeful) injected subcutaneously. For days the patient should take nothing but milk and ice; if inanition threatens, nutritive enemata should be ordered (vide page 33). When death from hemorrhage threatens, transfusion has been performed. But apart from the fact that transfusion of blood is no longer regarded as a rational procedure, Czerny and Kussmaul report a case of intestinal hemorrhage in which, after transfusion had been performed, renewed hemorrhage and death ensued. Transfusion with a solution of common salt is being more employed at the present time.

Caution should be exercised in the internal administration of styptics as they often produce vomiting. The following may be mentioned: \mathcal{R} Plumb. acetic., gr. $\frac{3}{4}$; opii, gr. $\frac{3}{16}$; every two hours.— \mathcal{R} Acid. tannic., gr. iss.; opii, gr. $\frac{3}{16}$; every two hours.— \mathcal{R} Liq. ferri sesquichlorat., 3 iij.; five to ten drops in water, repeated a number of times.— \mathcal{R} Seri lact. aluminat., \mathfrak{z} xvi.; one wineglassful every fifteen to thirty minutes.— \mathcal{R} Ol. terebinthin., five to ten drops in water, repeated several times.

Intermittent hæmatemesis should be treated with quinine (gr. xv.—xxx. daily in a single dose).

If symptoms of suffocation occur, the finger should be introduced into the introitus laryngis, in order to remove any clots which may be present.

The anæmic symptoms which are left over require nutritious, light diet, particularly in a fluid form. Iron preparations should be administered with caution, and only when gastric symptoms are no longer present.

2. *Acute Gastric Catarrh.*

(*Acute Gastritis.*)

I. ETIOLOGY.—Acute gastritis is sometimes primary, sometimes secondary to other diseases.

Errors in diet constitute the most frequent cause of acute primary gastritis. It is an every-day experience that overeating produces acute gastric catarrh, particularly if the individual usually eats sparingly. In such cases the gastric juice is too small in amount to digest the excess of food, and, in addition, the power of the muscular coat of the stomach is often too slight to propel the food at the proper period into the intestines. Hence fermentation of the food and irritation of the gastric mucous membrane.

Errors in the quality of the food may also produce the disease; for example, ingested foreign bodies (fruit pits, fish-bones, nails, hair), indigestible food (fatty articles, leguminous plants, particularly when they are not properly hulled), spoiled food (tainted meat, bad wine or beer, water from stagnant pools, etc.), unripe vegetables, and fruit.

Some individuals possess an idiosyncrasy with regard to certain articles of food, and their ingestion is always followed by acute gastritis, even when the patient is unaware of the nature of the food he has eaten.

Acute gastritis sometimes depends upon errors with regard to the

period of eating. Thus it has been observed as the result of prolonged fasting.

It may be produced by mechanical, thermal, and chemical irritants.

Among the mechanical irritants should be included hasty eating, in which the food is imperfectly masticated. This category also probably includes cases which have been reported as the result of eating cheese (Meschede), and raspberries with the larvæ of dipteræ (Gerhardt).

The disease may also be produced by the ingestion of very hot or very cold articles. A remarkable case of this kind is reported by Spry. While the watchman of a light-house was looking upwards at the burning of his tower some molten lead fell into his mouth, and then into the stomach. At his death, which occurred a few days later, the lead was found in the stomach, while the sole anatomical changes were those of gastritis.

Chemical irritants include chiefly the poisons (acids, alkalies, caustics, tartar emetic, etc.). Toxic gastritis also includes those forms which are observed in uræmia, cholæmia, gout, and alcoholic excesses.

Acute gastric catarrh may also be the result of a cold. Among the causes we must also mention injuries to the gastric region, as in lifting heavy loads, frequent and excessive straining.

A great influence is exerted by the nervous system, which probably acts by producing disturbances in the secretion and character of the gastric juice, thus interfering with digestion, and facilitating decomposition of the food. Thus many individuals suffer from acute gastritis after any violent emotion, or after coitus.

Acute gastritis sometimes occurs almost in an epidemic form. This is observed very often in midsummer, but also in the autumn and spring, when it is often associated with influenza. It is probably the result of infectious influences.

Primary acute gastritis is more frequent in men than in women. Catarrh of the gastric mucous membrane alone is more frequent in adults than in children, but gastro-intestinal catarrh is more frequent in the latter. The relation between the two diseases is readily understood. The imperfectly digested and partially decomposed food which passes from the stomach into the intestines produces inflammation of the mucous membrane of the latter.

Certain individuals present a special tendency to gastric catarrh. This is observed in conditions of anæmia and convalescence, in chlorosis, phthisis, syphilis, cancer, hysteria. Very slight causes will produce acute gastritis when the circulation is impaired in consequence of diseases of the heart, lungs, or liver. In some cases there is an hereditary predisposition to the disease.

Secondary gastritis is observed in many febrile and infectious diseases. It sometimes opens the scene, and in children it not infrequently takes the place of the initial chill. It is more frequent in some infectious diseases than in others. It is an almost constant prodrome of scarlatina, and is also very frequent in erysipelas.

Acute gastritis develops not infrequently after affections of the mouth, salivary glands, and pharynx. For example, in salivation when large amounts of saliva are swallowed, or when putrid, purulent secretion is swallowed in affections of the pharynx. It is observed also in putrid bronchitis and pulmonary gangrene, more rarely in pulmonary abscess or in empyema which has ruptured into the lungs.

In rare cases, acute gastritis is propagated from the intestines, but it more frequently accompanies peritonitis.

II. ANATOMICAL CHANGES.—Acute gastritis is rarely fatal, so that our knowledge of the anatomical lesions is based partly on theory, partly on experiments. The pyloric part of the stomach is affected chiefly or exclusively. The mucous membrane is unusually red, loosened, swollen, and covered with vitreous or slightly cloudy mucus. This is usually removed with difficulty. The redness may be uniform, in patches, or arborescent. Small extravasations are observed occasionally, and the mucous masses may be streaked with blood.

Some of these phenomena, especially the hyperæmia, may disappear in part after death. On the other hand, physiological hyperæmia may be found in autopsies on individuals who died suddenly during digestion. Furthermore, catarrhal swelling should not be mistaken for cadaverous softening and maceration.

Ebstein produced gastritis experimentally by feeding starving dogs with whiskey. On microscopical examination, the simple and compound pepsin glands presented appearances similar to those observed during digestion. The peptic cells were not appreciably changed, the mucous cells were opaque, granular, shrunken, partly fatty and stained very deep by reagents. The cylindrical cells were in a mucoid condition. Numerous round cells were accumulated in the interglandular connective tissue. In addition, Ebstein found dilatation of the blood-vessels, swelling of the endothelium of the lymphatics, proliferation of the nuclei and desquamation.

III. SYMPTOMS AND DIAGNOSIS.—Anorexia is the chief symptom of acute gastritis. The mere thought or sight of food often produces nausea. Many patients have a longing for highly-spiced, pungent articles of food. Thirst is almost always increased.

Nausea and vomiting are observed almost constantly. The vomited matters consist at first of partly unchanged, partly decomposed food. Later, we find a tough, stringy, mucoid fluid, which is vomited with difficulty. It is sometimes streaked with blood. Finally, the patients vomit a yellow, greenish-gray, or greenish-black biliary fluid which has an intensely bitter taste. The act of vomiting is very often preceded by a sort of "imperative idea." The mind is continually recurring to certain articles of food, the conception of which intensifies the nausea.

Many patients are very much annoyed by singultus. The gases discharged are sometimes odorless, sometimes sour-smelling, or they have a sulphuretted hydrogen stench, or an indefinable nauseous odor. Rancid gases, or very acid gastric contents, may give rise to a burning feeling (heart-burn, pyrosis) in the stomach, or in the œsophagus (after vomiting).

The patients generally complain of fulness and distention of the epigastrium. There is sometimes a feeling of pressure and even pain, which may be confined to the epigastrium, or may radiate towards the spine and scapulæ. In some cases, pain is produced by the ingestion of food.

As a rule, the tongue is coated. There is not infrequently a stale, pasty taste in the mouth, with increased secretion of saliva and fœtor ex ore. Herpes (small, light-yellow vesicles which dry into brownish-yellow crusts) sometimes forms upon the lips.

The bowels are almost always constipated. Diarrhœa occurs only when the inflammation extends to the intestinal mucous membrane.

The urine is usually diminished in quantity. It has a dark color, and, on cooling, often deposits a bright-red sediment of urates. In one case, Senator noticed an odor of sulphuretted hydrogen in the sediment.

Nervous disturbances are often very noticeable. Complaints are

made of a feeling of dulness in the head, abnormal sensations of pressure, throbbing in the frontal or occipital region. The patients fall into a melancholy mood, and sometimes feel so miserable that they are apprehensive of a serious illness. Their thoughts are often confused, and there is often an inability to perform any mental effort. A feeling of vertigo is often experienced, and is sometimes so severe that the patient is unable to stand. Præcordial terror and palpitation of the heart occur occasionally.

The temperature may be normal, or there is a slight rise. In some cases, the fever is extremely high, and delirium and eclampsia may develop, particularly in children. If the fever continues for some days, it may be mistaken for typhoid fever. This is differentiated by the demonstration of an error of diet, or other obvious cause, the usually sudden onset, unusual course of the fever, and the more rapid and sudden termination.

Acute gastritis may last from a few hours to several days, or even two weeks. One attack predisposes to a relapse, and if repeated attacks occur, the condition may become chronic.

IV. PROGNOSIS.—The prognosis is favorable, except in those cases in which the cause produces an irreparable lesion.

V. TREATMENT.—Rational diet will often prevent the development of acute gastritis.

After the disease has developed, the causal factors should first be considered.

Emetics are indicated when the stomach is overloaded; for example, apomorphine (gr. iss. : 3 iij., $\frac{1}{4}$ – $\frac{1}{2}$ syringeful subcutaneously). The combination of ipecacuanha (gr. vij.) with tartar emetic (gr. $\frac{3}{4}$) (one powder every ten minutes until emesis is produced) is less serviceable, because it irritates the gastric mucous membrane. Ipecacuanha alone (gr. vij. every ten minutes) should only be ordered in children.

If the stomach is distended with gas, and the patients complain of eructations of irritating gases, we may order a few glasses of selters water; or sodium bicarb., sodium salicylic., āā 3 i. (as much as can be placed on the tip of a knife, every two hours); or sodium bicarb., gr. vij.; resorcin, gr. iss. (one powder every two hours).

Laxatives may be recommended if we suspect that the decomposed gastric contents have entered the intestines. For example, calomel and jalap, āā gr. v.; or magnes. ust., 3 iij.; aq. destill., q. s. ad $\frac{3}{4}$ vij. M. D. S. one tablespoonful every hour; or pulv. liq. comp., two tablespoonfuls in water.

Violent gastric pains are relieved by warm poultices and injections of morphine in the region of the stomach. The latter are also useful when vomiting occurs from excessive peristalsis of the stomach.

But if there are no special indications, we may confine ourselves to dietetic measures alone. For one or more days the patient should be restricted to water, soup, or weak meat soup, weak tea, good wine, or water; at the most boiled milk. In addition, acid. hydrochlor. dil., gtt. v., in one-half wineglassful of lukewarm water t. i. d., half an hour after meals.

3. Chronic Gastritis.

I. ETIOLOGY.—Chronic gastritis either develops from a relapsing acute catarrh, or it is chronic from the beginning.

It is remarkably frequent in drunkards, particularly in those who

imbibe alcohol in the purest form possible. No small proportion of drunkards fall a prey to chronic gastritis, while others perish from degeneration of the heart muscle, chronic inflammation of the liver and kidneys, or pneumonia.

The disease is very often the result of irrational diet, the result of irregularity of the meals, or excessive haste in eating. This is especially true of certain professions, such as medicine and law. Imperfect teeth also predispose to chronic gastritis. It is often found among the poor, whose chief nutriment consists of vegetables which must be taken in large amounts to meet the needs of the body. In this case over-burdening of the stomach and easily decomposed food are the two chief injurious factors.

The disease is common in other protracted gastric affections, for example, round ulcer and carcinoma. Indeed, the symptoms of gastritis may entirely conceal those of the primary affection.

Chronic gastritis is also the result of circulatory disturbances. It occurs in diseases of the portal vein, in many hepatic affections, particularly cirrhosis, and in all conditions which interfere with the outflow of blood from the inferior vena cava (valvular diseases, changes in the heart muscle, pulmonary emphysema, etc.).

It often develops in certain constitutional diseases, such as anæmia, chlorosis, phthisis, cancer, syphilis, Bright's disease, etc. In such cases also the gastric symptoms may occupy the most prominent part.

It is more frequent in adults, particularly in males, and is found particularly among those who follow sedentary occupations.

II. ANATOMICAL CHANGES.—Abnormal color and swelling of the mucous membrane and excessive secretion are the most important changes. They affect chiefly the pyloric portion.

The mucous membrane is usually brownish-red or grayish-red. A few large and unusually dilated vessels are often seen beneath the mucous membrane. Sometimes we find scattered extravasations of blood and superficial losses of substance. If the catarrh has lasted for some time, the mucous membrane assumes a grayish-black or slate-gray color. This is the result of metamorphosis of the pigment of the extravasated blood-corpuscles, and its deposition in the interglandular tissue, the cells of the glands, and mucous membrane. In rarer cases the mucous membrane is unusually pale.

The loosening and swelling of the membrane are often marked. The surface is covered with thick masses of mucus, sometimes vitreous and translucent, sometimes cloudy and slightly purulent, sometimes tinged with blood or colored gray or brownish.

Chronic gastritis also gives rise in many cases to thickening from inflammatory hyperplasia, affecting chiefly the interglandular connective tissue. The compression produces atrophy of numerous glands. In addition, their long diameter is often shortened (so-called atrophic pigment induration).

The submucous and muscular layers often taken part in the inflammatory hyperplasia. The muscular fibres may increase in size to such an extent that the muscular layer attains a thickness of two cm. This increase in thickness is generally confined to certain parts of the muscular tunic. If it is confined to the pylorus it may give rise to stenosis, which in its turn causes stagnation and decomposition of food and dilatation of the stomach.

Even the serous layer is occasionally thickened; in places it is opaque and rough.

The surface of the mucous membrane is very often uneven and nodular. Sometimes there are numerous large prominences, sometimes the surface has a finely nodular appearance (*état mamellonné*).

This condition is sometimes produced by submucous accumulations of fat, or inflammatory hyperplasia of the lymph follicles. Ebstein attaches importance to inflammatory hypertrophy of the interglandular connective tissue. In some cases Jones found partial atrophy and fissures in the mucous membrane, in others he attributes the condition to excessive contraction of the smooth muscular fibres. Rindfleisch believes that the surface has grown so large that it can no longer be spread in a uniform layer over the inner surface of the stomach. Ziegler describes hyperplasia of the submucosa in the prominent parts of the mucous membrane.

Secondary changes sometimes develop. These include cystoid degeneration of some of the glands from occlusion of the excretory duct, and an accumulation of mucoid fluid within the gland. The mucous membrane is sometimes elongated into pendulous outgrowths (so-called polypi of the stomach). As many as thirty may be present. If situated at the gastric orifices they may produce symptoms of stenosis. Less prominent, but more numerous proliferations give rise to that form of the disease which has been described as gastritis proliferata or verrucosa.

III. SYMPTOMS.—The symptoms are almost identical with those of acute gastritis, but they are less severe. The majority of patients complain of a feeling of fulness and tenderness in the epigastrium, sometimes intensified to violent pain.

The patients suffer from loss of appetite, but boulimia may be noticed at irregular intervals. There is a frequent desire for strongly spiced articles of food. Thirst is sometimes increased, but not so often as in acute gastritis. Vomiting is also less frequent.

The vomited matters are sometimes unchanged, sometimes in a condition of fermentation. Bilious vomiting is also observed. Yeast fungus and sarcina ventriculi are found not infrequently with the microscope.

Morning sickness often occurs in drunkards. The vomited matter is mucous and stringy, usually alkaline and consists chiefly of saliva which is excreted in increased amount on account of reflex irritation of the nerves of the salivary glands, and is swallowed during the night. The patients sometimes vomit peculiarly tough, vitreous, gum-like masses, which are the result of mucous fermentation of the hydrocarbons.

There are frequent eructations of odorless or rancid gases, and sometimes a small amount of the gastric contents passes into the mouth at the same time. This is associated not infrequently with a feeling of great relief. Heart-burn (pyrosis) very often persists for hours afterwards.

The tongue may be entirely clear, but, as a rule, it has a thick, gray, yellowish or brownish coating, and its edges contain the impressions of the teeth. The saliva is generally increased in amount. *Fœtor ex ore* is often observed. The patients often complain of a stale, foul taste in the mouth.

The gastric region may be distended, and is often sensitive on pressure, most markedly in the region of the pylorus.

The urine is scanty, high-colored, often deposits a brick-red sediment

on cooling, and contains an excessive amount of oxalate of lime and phosphates.

The bowels are usually constipated unless enteritis is also present. In the latter event, diarrhœa may alternate with constipation.

The patients complain not infrequently of palpitation and præcordial terror which, according to French writers, may give rise to dilatation of the right ventricle. It is assumed that reflex irritation from the stomach causes contraction of the pulmonary vessels in the tracts of the vagus and sympathetic, and that this causes increased pressure in the pulmonary artery. Stenocardic and asthmatic symptoms are occasionally observed.

The patients often feel as if they are suffering from some severe disease. They put no trust in their mental or physical powers, and regard their condition as hopeless.

Many suffer from vertigo (*vertigo e stomacho læso*), and this seems to confirm their suspicions of an incurable disease of the brain or spinal cord. The dizziness is often felt only in the morning. In one case, Leube was able to produce this symptom by pressing upon the patient's stomach while in the recumbent position, and then allowing him to stand up.

If the disease lasts for a long time, the general nutrition becomes impaired. The signs of dissolution of the blood sometimes develop, and numerous hemorrhages appear upon the skin and mucous membranes. Eczema and urticaria appear occasionally. If the gastric catarrh is secondary to a serious affection, death may ensue as the result of increasing marasmus.

The disease lasts for months or years, with frequent remissions and exacerbations.

IV. DIAGNOSIS.—It is often difficult to decide whether chronic gastritis is an independent affection or is dependent on cancer or ulcer of the stomach.

Hypertrophy of the walls of the stomach may develop to such an extent as the result of catarrh that the thickened greater curvature or pylorus may be felt on palpation, but in such cases the prominence is smooth. The age of the patient and the long continuance of gastric symptoms without cachexia may also be useful in differential diagnosis from cancer. The absence or infrequency of vomiting favors the diagnosis of simple catarrh. According to Bamberger, an abundance of *sarcina ventriculi* in the vomited matters is indicative of cancer.

When a gastric tumor is not demonstrable, a cancer may be overlooked. Our suspicions should be aroused by the advanced age of the patient, cachexia, swelling of the left supraclavicular glands and œdema.

Ulcer of the stomach often affects anæmic or chlorotic individuals, the pain is more violent and localized than in simple gastritis, and follows more closely on the ingestion of food. Hæmatemesis would at once decide in favor of ulcer.

V. PROGNOSIS.—The prognosis is bad when the gastritis is secondary to some incurable affection. In the primary variety, recovery can be looked for only when the patients can break their bad habits and adhere strictly to the prescribed diet.

VI. TREATMENT.—Prophylactic measures possess no slight importance. We refer to proper mastication of the food, proper intervals between meals, intact condition of the teeth, etc. Individuals who are liable to gastritis from cold should wear a warm belly band.

Dietetic measures constitute the chief feature in treatment. Leube's dietary contains four groups, arranged, according to their digestibility, in the following order: *a.* Meat soup, Leube-Rosenthal's meat solution, milk, raw and very soft boiled eggs. *b.* Stewed calves' brain, thymus gland of the calf, chicken, pigeon, calves' feet, in addition glutinous soup at dinner, and porridge at supper. *c.* Boiled scraped beef and raw ham. *d.* Boiled chicken, pigeon, rare roast beef, veal, hare, pike, trout (with caution), macaroni, bouillon with rice, and small amounts of old white or red wine, or Bavarian beer. We should carefully prescribe the amount of food to be taken, and the hours of eating; the patient should eat slowly, and masticate thoroughly. Fresh bread must be forbidden. It should be toasted, or we may order zwieback or English crackers (Albert). Fatty articles, fresh vegetables, and amylaceous articles are tabooed.

Krukenberg recommended a milk or butter-milk cure, but in some patients this produces disgust or violent heart-burn. The latter may sometimes be prevented by the addition of lime-water or bicarbonate of soda.

Some patients suffer less from a diet of more spicy articles, for example, smoked tongue or beef, pork sausage.

In chlorotic and anæmic patients, the careful use of hydrochloric acid is often serviceable. It is sufficient to give five drops in half a wine-glassful of lukewarm water half an hour after dinner. Its action is sometimes increased by the addition of a little pepsin.

When fermentation of the contents of the stomach occurs to a slight degree, we may resort to the internal administration of salicylic acid (gr. viij. t. i. d., half hour after meals), resorcin (gr. iss. t. i. d.), creasote (gtt. v. in 15 pills, one pill t. i. d.), or benzolum (twenty drops twice a day).

There are two indications for the use of the stomach-pump in chronic gastritis, viz., excessive fermentation of the gastric contents, and inability of the organ to propel its contents. The pump should then be used every morning until the symptoms of fermentation and stasis are no longer demonstrable. The stomach should be washed with lukewarm water (30 to 35° C.) until it runs out perfectly clear. It is advisable to follow this up with a weak solution (1 to 2 per cent) of natrium salicylicum, or resorcin, aqua creasoti (1 : 2 of water), benzolum ($\frac{1}{2}$ per cent). If there is an excessive formation of acid, a solution of sodium bicarbonate (1 to 2 per cent) or Carlsbad salts (1 to 2 per cent) is indicated.

If we have reason to suspect impaired muscular tonus of the organ, we may order bitters, for example, strychnia (gr. ss., pulv. althææ, q.s. ut f. pil. No. 15. D. S. 1 pill t. i. d.), tinct. amara (25 to 30 drops t. i. d.), tinct. gentian, 25 to 30 drops t. i. d., tinct. quassia, cortex condurango, tinct. quiniæ comp., etc. Cold baths and rubbings, sea baths, and cold-water cures are often useful under such circumstances. Electrical applications are also said to be attended with good results. One electrode of a strong faradic current is placed upon the back, the other is passed slowly across the epigastrium from left to right, or a flexible stomach electrode is introduced into the stomach, and the other one placed upon the epigastrium.

Bismuth, nitrate of silver, or opium must be administered with caution, as they sometimes aggravate the symptoms.

Certain symptoms may require special treatment. If the pain is very

severe, we may order: \mathcal{R} Aq. amygdal. amar. 3 iij., morphin. hydrochlor. gr. iss. M. D. S. 10 drops t. i. d.; extract. belladonn. gr. $\frac{1}{4}$ t. i. d.; morphin. hydrochlor. gr. $\frac{1}{2}$ t. i. d.; or \mathcal{R} Chloral. hydrat. gr. lxxv., pulv. althææ et tragacanth. q. s. ut f. pil. No. xx. D. S. One pill t. i. d. before meals.

Heartburn may be relieved by magnesia usta, natrium bicarbonate, aq. calcis, or the previously mentioned antifermentatives.

Constipation may be combated by: \mathcal{R} Aloes, ext. rhei co., jalapæ aa gr. xv., pulv. et suc. liq. q. s. ut f. pil. No. 30. D. S. 2 to 4 pills at night, etc.

When the gastritis is secondary to diseases of the circulatory or respiratory apparatus or liver, our chief attention should be directed to the primary disease.

Special repute in the treatment of chronic gastritis is enjoyed by mineral waters, particularly the alkaline acidulous waters (chief constituents: carbonic acid and natrium carbonate), alkaline-muriatic waters (chief constituents: carbonic acid, sodium carbonate, and sodium chloride), alkaline-saline waters (large amount of sodium sulphate) and salt water.

a. The alkaline acidulous wells are preferable when the patients suffer frequently from eructations, heartburn, and gastric pain. *b.* The alkaline-muriatic waters are indicated when there is marked nausea, with a flat taste in the mouth, and lively secretion of tough mucus. *c.* The alkaline-saline waters are best in obstinate constipation (Carlsbad is particularly indicated if the stomach is very sensitive to pressure and spicy articles are poorly tolerated, while Marienbad is useful in obese individuals and drunkards). *d.* Salt waters are specially useful in very feeble individuals.

Ferruginous waters are indicated when the patients are anæmic.

4. Purulent Gastritis.

(*Gastritis phlegmonosa s. submucosa.*)

I. ANATOMICAL CHANGES.—Purulent gastritis involves exclusively or mainly the submucous cellular tissue, either as a diffuse infiltration or circumscribed abscess.

In diffuse purulent infiltration the submucosa may attain a thickness of one cm. The tissue often has a gelatinous look, and is infiltrated with puriform fluid, which may contain an excess of serum or fibrin, or be purely purulent. The infiltration may sometimes be squeezed out of the tissue. The pus sometimes perforates the mucous membrane in many places. The openings vary in size from that of a pin's head to that of a bean. The larger ones are irregular in shape, the small ones are round. Upon the introduction of a sound, the mucous membrane is often found to be separated from the submucosa over a considerable area. As a rule, these changes are most marked near the pylorus. In Chvostek's case, they extended to the œsophagus.

The mucous membrane is unchanged in some cases, and is even very pale in color. In a case reported by Glax, it formed a fluctuating mass, which could be moved to and fro upon its base. In some cases it is injected in patches, in others diffusely inflamed. Rokitansky reported one case in which the mucous membrane was converted into a pulpy, blackish-brown necrotic mass.

From the submucosa the pus passes along the intermuscular connec-

tive tissue septa into the muscular coat and subserous cellular tissue. The muscular coat may be almost entirely destroyed. Peritonitis is observed in almost all cases of phlegmonous gastritis. Key and Malmsten observed marked distention and sinuosity of the subperitoneal lymphatics. The stomach is generally distended with gas, and contains a bile-stained or brownish, flocculent, opaque fluid.

The following lesions have also been observed occasionally: ulceration, cancer, enlargement of the spleen, acute and chronic nephritis, diphtheria of the large intestine, gall-stones, gangrene, and perforation of the gall-bladder, pleurisy, mediastinitis, pericarditis.

Abscess of the stomach is a circumscribed accumulation of pus in the submucosa, and may attain the size of a fist. Several abscesses are sometimes present. The pus may burst into the cavity of the stomach, the peritoneum or adjacent organs.

II. ETIOLOGY.—The disease may be primary or secondary.

The causes of primary phlegmonous gastritis cannot always be determined. Cold, injury, errors of diet have been mentioned, but a certain predisposition seems to be necessary to its development. This may be the result of the abuse of alcohol.

Secondary phlegmonous gastritis develops during certain infectious diseases, especially typhoid fever, variola, pyæmia, puerperal fever, peritonitis, and splenic fever. It may also follow the ingestion of caustic poisons.

It is more frequent in men than in women (among 45 cases, 38 occurred in men, 7 in women). As a rule, it develops in middle life. The youngest patient was 17 years old, the oldest 76 years.

III. SYMPTOMS.—The disease either begins suddenly, or is preceded for a few days by anorexia, vomiting, and a feeling of pressure in the epigastrium. In secondary gastritis, symptoms may be entirely absent, or, at least, overlooked on account of the severity of the primary disease.

The patients usually complain of pain in the gastric region, sometimes extending to the right or left hypochondrium. There is often no increase of pain on pressure. Vomiting or eructation is almost always present. The vomited matters consist of the contents of the stomach, or are biliary or brownish; pus is rarely vomited. There is usually complete anorexia with almost unquenchable thirst. The bowels are sometimes constipated, sometimes the passages are diarrhœal, occasionally bloody; in Key and Malmstens' case, they were dysenteric in character. In this case, the urine was bloody and contained casts; it is generally scanty and concentrated. Physical examination is negative in many cases, in others a circumscribed abscess can be felt as a tumor.

The general condition is seriously affected in the majority of cases. The temperature may reach 41° C., and a typhoid condition is often observed, and may be mistaken for typhoid fever or meningitis. In other cases, the abdomen is distended and sensitive, and the signs of peritonitis develop.

Ap pyrexial cases are rare, but death sometimes occurs unexpectedly without any noteworthy previous symptoms.

The disease usually runs an acute course. As a rule, death occurs inside of two weeks, not infrequently in a few hours or days. The disease rarely extends over several weeks or even months.

IV. DIAGNOSIS.—This is hardly possible unless pus is vomited. In

such cases, we must endeavor to satisfy ourselves that the pus has not perforated the stomach from the pleura, pericardium, liver, spleen, kidneys, peritoneum, or spinal column. The vomited matters should also be examined with the microscope, since the pus may not be visible to the naked eye. The disease may be mistaken for typhoid fever, meningitis, or peritonitis; in other cases, it remains latent during life.

V. PROGNOSIS.—The prognosis is usually unfavorable. Brand and Dittrich showed that recovery is possible, since they found cicatricial tissue in parts of the submucosa, which had produced stenosis in the pyloric region. Cases have been reported of recovery from abscess of the stomach after the vomiting of pus.

VI. TREATMENT.—This is purely symptomatic. Vomiting and pain are combated by subcutaneous injections of morphine, an ice-bag to the stomach, and ingestion of pieces of ice. Rectal alimentation should be resorted to (vide page 32). Fever is treated with antipyrin (3 i. to iss. in $\frac{5}{8}$ ij. of lukewarm water per rectum). In collapse, stimulants are administered, preferably camphor gr. xv., ol. amygdal. 3 iij., one syringe-ful three or four times a day.

5. Toxic Gastritis.

(*Gastritis venenata.*)

I. ETIOLOGY.—This is generally the result of the ingestion of acids or alkalies, but may also be produced by any other irritating substance, and by certain vegetable poisons.

II. ANATOMICAL CHANGES.—These depend mainly on the amount and concentration of the poison which has entered the stomach. The most extensive lesions are generally found in cases of suicide. When the poison is taken accidentally, the patient soon discovers the mistake, and attempts to get rid of the poison as soon as possible. Under such circumstances, the changes may be confined to the mouth and œsophagus.

The nature of the poison often exerts an influence on the character of the gastric lesions. In sulphuric-acid poisoning, the scurf upon the mucous membrane is grayish-black; in nitric-acid poisoning, it is yellowish (xanthoprotein); in poisoning with alkalies, it is brownish; in copper-poisoning, it has a blue or green color (the latter changing to dark-blue on the addition of ammonia); in silver-poisoning, it is deep-black. In cases of phosphorus-poisoning, the mucous membrane not infrequently has a milky, opaque, or yellowish appearance. The glands of the mucous membrane are in a state of marked fatty degeneration (so-called gastritis parenchymatosa, s. glandularis, s. gastro-adenitis).

The lesions are most marked in those places with which the poison has been longest in contact (fundus and posterior wall). In mild cases, branching stripes are sometimes seen running along the posterior wall from the cardiac end to the fundus, where they attain greater dimensions.

According to the intensity of the irritant action, we will find superficial excoriations, removal of the epithelial layer, simple or hemorrhagic catarrh (rarely croupous inflammation in ammonia-poisoning, or the formation of pustules in poisoning with tartar emetic), or the formation of a scurf, while the other coats are infiltrated with serum, or all the layers of the stomach are seriously implicated. In the most severe cases, the stomach forms a black, sloughy mass, which tears on the slightest touch, or has ruptured spontaneously, and has poured its brownish or

hemorrhagic contents into the abdominal cavity. The vessels of the stomach contain firm clots, or dark, fluid, tar-like blood.

The lesions sometimes extend, in a less marked degree, to the mucous membrane of the intestines.

Lesions which affect chiefly the mucous membrane may recover. After suppuration, the slough is thrown off, and cicatricial connective tissue develops. Large pieces of the mucous membrane are sometimes vomited.

Cicatrization may give rise to new dangers by causing stenosis of the entire gastric cavity, or of the orifices. It is said that the organ sometimes contracts to the dimensions of a hen's egg.

III. SYMPTOMS.—The first symptom is burning, agonizing pain in the region of the stomach, which is increased on the slightest pressure. The patients toss to and fro restlessly and anxiously. As a rule, pain is also felt along the spine (œsophagitis) and in the mouth and pharynx.

Singultus develops, with occasional vomiting, usually of bloody masses. The thirst is unquenchable. The passages are often diarrhœal and mixed with blood.

If peritonitis develops, the abdomen becomes tympanitic, tender on pressure, and presents abnormal areas of dulness. In perforation-peritonitis, the hepatic dulness disappears, because the air which escapes from the stomach pushes the liver away from the wall of the thorax.

As a rule, there are very grave disturbances of the general condition. The sensorium is often clouded. The pulse is small and unusually frequent. The skin is cold and covered with clammy sweat. Bamberger observed two deaths from shock, although the stomach did not present any marked lesions.

Death may occur within a few hours. In other cases, cicatrization occurs, but recovery is sometimes only temporary, and the patient dies from inanition. Profuse and occasionally fatal hemorrhage may occur when the slough is thrown off.

IV. DIAGNOSIS.—That we have to deal with poisoning must be concluded from the usually sudden illness. The nature of the poison is determined, as a rule, by the history of the case. Whether we have to deal, in doubtful cases, with poisoning with alkalies or acids is shown by the reaction of the buccal mucous membrane to litmus paper.

V. PROGNOSIS.—The prognosis is always grave, and should be guarded for a long time, in view of the possibility of hemorrhage or retraction of the cicatricial tissue.

VI. TREATMENT.—In recent cases of poisoning with acids, an alkali should be administered, such as *magnesia usta* (gr. lxxv. to a glass of milk with ice, a tablespoonful every ten minutes), or, in case of necessity, powdered chalk or lime. In poisoning with alkalies, dilute vinegar may be administered. In other cases, the stomach should be washed out through a soft tube, and then the ordinary antidotes given.

Pieces of ice, ice and milk, or an ice-bag to the epigastrium may be ordered for the gastritis. Violent pain should be relieved by subcutaneous injections of morphine.

6. Round Ulcer of the Stomach.

(*Ulcus ventriculi simplex s. chronicum s. perforans, s. pepticum.*)

I. ANATOMICAL CHANGES.—The round gastric ulcer (either recent

or cicatrized) is found with remarkable frequency (in about five per cent of all autopsies).

As a rule, the ulcer forms a perfectly round or elongated, oval loss of substance. This is particularly true of small ulcers. Large ones are irregular, either because the ulcer has spread in different directions with varying rapidity, or because adjacent losses of substance have coalesced with one another. In some cases the diameter of the ulcer is hardly one centimetre, in others it may attain the size of the palm of the hand. Cruveilhier described a case in which the ulceration extended along the lesser curvature from the pylorus to the cardiac extremity. It is situated most frequently in the pyloric portion upon the posterior wall near the lesser curvature, most infrequently at the fundus.

Brinton collated the following statistics :

Posterior wall,	86 (42 per cent).
Lesser curvature,	55 (26.8 ").
Pylorus,	32 (15.6 ").
Anterior and posterior walls,	13 (6.3 ").
Anterior wall,	10 (4.9 ").
Greater curvature,	5 (2.4 ").
Cardia,	4 (2.0 ").

205

There is usually but one ulcer, more rarely several: even as many as eight have been observed in different parts of the organ. As a rule, they present various stages of development. The round ulcer has also been found in the duodenum and œsophagus.

The ulcer has such sharply defined borders that it very often looks as if it had been punched out. At first the loss of substance affects only the mucous membrane, then it spreads to the muscular coat, and finally to the serous layer. It is always funnel-shaped, the small end being situated at the serous layer. But the centre of the loss of substance in the mucous membrane and the tip of the funnel are not situated directly above one another. In ulcers of the upper half of the stomach the tip is usually directed upwards, in those of the lower half downwards, corresponding to the mode of ramification of the gastric vessels. The edges of the ulcer are rarely thickened, and an inflammatory zone is almost always absent. The edges are terrace-shaped, their slope being steeper in the cardiac region than near the pylorus.

The base of the ulcer is remarkably clean in many cases. In others it has a brownish, hemorrhagic coating.

In many cases perforation of the stomach is prevented by adhesive peritonitis which binds the stomach to adjacent organs. In most cases we find adhesions to the pancreas and adjacent lymphatic glands, to the left lobe of the liver or the omentum, more rarely to the spleen, colon, small intestine, diaphragm or anterior abdominal wall. Sacculated peritonitis sometimes develops at the site of threatened perforation, and the rupture occurs into the sacculated peritoneal space. The round ulcer may give rise to subphrenic pyopneumothorax (Vol. I., page 375).

If the base of the ulcer is formed by the adherent pancreas, liver, or spleen, the mucous membrane, as a rule, projects over the muscular coat and forms the transition from the cavity of the stomach to the base of the ulcer. The adherent organs are sometimes involved in the destructive process, so that the latter may produce large cavities filled with pus or

ichor. In this manner the stomach may connect with other cavities and organs (internal gastric fistula), such as the duodenum, colon, pleural or pericardial cavities, left ventricle, or through the lungs into the bronchi, gall-bladder. If adhesions to the anterior abdominal wall have formed, an external gastric fistula may develop, the complete formation of which may be preceded by emphysema of the skin.

Perforation into the peritoneal cavity depends chiefly on two factors: the rapidity with which the ulcer forms, and the more or less favorable opportunity for the production of adhesions. Ulcers of the anterior wall are especially apt to lead to perforation because the mobility of this part is not favorable to the production of adhesions. Among 75 ulcers of the anterior wall, perforation occurred 64 times; among 30 cases of the cardiac extremity, it occurred twelve times.

The development of the ulcer within the walls of the stomach and its extension to adjacent organs afford the possibility for hemorrhage. On account of its course along the posterior wall of the stomach (the most frequent site of ulceration) the *arteria lienalis* is most frequently eroded. The bleeding artery is sometimes found in a condition of aneurismal dilatation. Audral observed a fatal hemorrhage from varicose veins near the ulcer. In order to detect the bleeding vessel, the main trunk should be injected with water; this will escape from the erosion.

Cicatrization of gastric ulcers is not uncommon. If the loss of substance is slight and affects only the mucous membrane, the cicatrization may be so complete as to be readily overlooked on autopsy. If the ulcer is deeper and larger, the adjacent mucous membrane is involved in the cicatricial process. Thick, fibrous callosities sometimes form, and their retraction may impart an unusual shape to the organ. Ulcers upon the posterior wall may spread in a girdle around the stomach, and their subsequent cicatrization may divide the organ into abnormal cavities (hour-glass shape). These are most marked if the curvatures of the stomach are specially involved in the retracting process. Cases have been observed in which the anterior division was alone employed in digestion, and was connected by a fistula with the colon or duodenum.

Another mode of recovery is the formation of numerous fibrous adhesions: the stomach is sometimes adherent to all the adjacent organs. The adhesions may also be the cause of death. In one case the adhesions between the stomach and gall-bladder ruptured and produced fatal hemorrhage.

II. ETIOLOGY.—The disease is extremely rare before the age of 14 years, but cases have been reported at the age of 3, 3½, and 4 years. Vergely observed a recent gastric ulcer in a woman *æt.* 83 years. Clinical observation teaches that the disease is most common from the 15th to the 30th years.

Women are attacked more frequently than men. Brinton found a proportion of 2 : 1; With of 7.3 : 1. Starcke observed the disease more frequently in men.

A marked predisposition is offered by certain general diseases, such as chlorosis. This is also true of phthisis, arterio-sclerosis, and syphilis. According to Rokitansky it may be associated with intermittent fever. London reported a case which he attributed to pigment embolism of the gastric vessels in protracted intermittent fever. Among other causes may be mentioned abuse of alcohol, indigestible food, inordinate ingestion of vegetables, and imperfect mastication.

Bamberger noticed the disease frequently in cooks. Speck attributes

its extraordinary frequency in Eastern Siberia to the almost exclusive diet of fatty fish.

Violent vomiting, severe gastritis, injury of the mucous membrane by a blow or fall, or by poisonous or foreign bodies, may give rise to hemorrhage into the mucous membrane and then to ulceration. After extensive cutaneous burns, ulceration of the stomach and duodenum has been observed. This is probably the result of destruction of red blood-globules and occlusion of the vessels of the mucous membrane by the products of corpuscular degeneration.

Certain trades exert an influence on the development of round gastric ulcer. Bernutz calls attention to its frequency in potters and glass workers, and explains it by the ingestion of sharp particles of dust. I have observed the disease frequently in metal workers.

The shape of the ulcers resembles the mode of ramification of the gastric vessels, and it often seems as if the subdivisions of the smaller arteries were to a certain extent punched out of the mucous membrane. In addition, the round ulcer develops under circumstances which warrant us in assuming changes in the walls of the vessels and circulatory disturbances in general. It is known that chlorosis leads to fatty degeneration of the vessels. This is also true of pulmonary phthisis which, in addition, may give rise, like syphilis, to waxy degeneration of the vessels. Atheromatous changes in the vessels have also been observed.

The influence of the acid gastric juice is evident from the fact that round ulcers are found only in those parts of the digestive tract in which its digestive effect is present.

Under normal conditions, the gastric juice does not digest the wall of the stomach because it is neutralized by the alkaline blood. Self-digestion is possible if the gastric juice is excessively acid, and is no longer completely neutralized by the blood, or if the circulation in the mucous membrane is so feeble that neutralization is impossible. The former view is not plausible because the ulcer would then extend over large surfaces.

As a matter of course, various circulatory disturbances may give rise to a gastric ulcer; there must simply be a disproportion in the process of neutralization. The more marked this is, the more rapidly will the ulcer develop. It may result from embolism, thrombosis, simple stenosis following arterio-sclerosis, deep-spreading ecchymoses. It is doubtful, however, whether mere spasm of the arteries leads to the formation of an ulcer. Axel Key believes that spasm of the muscular coat of the stomach causes disturbance of the venous circulation, and thus may give rise to an ulcer. Boettcher attributes gastric ulcers to the action of schizomycetes.

Ulcers have been produced experimentally by ligature or embolism of the gastric vessels, ligature of the portal vein, by injuries of the central nervous system which caused hemorrhages into the mucous membrane of the stomach.

III. SYMPTOMS.—In a few cases the development of gastric ulcer is entirely latent. Individuals who were previously healthy, or had merely suffered for a few days from trifling gastric symptoms, are suddenly seized with a profuse gastric hemorrhage, or suddenly fall down, complain of intolerable pain in the abdomen or that something has burst, and die in a few hours or in one to three days, with symptoms of perforation-peritonitis.

In the large majority of cases, annoying symptoms develop, among which pain in the stomach is most constant. As a rule, it begins after eating, either immediately or within one to two hours. It is so much more severe, the larger the meal and the more imperfect the mastication of the food.

In rare cases the pain occurs in the morning or while fasting, and disappears after eating. It is usually described as boring, gnawing, burning, more rarely as lancinating. It is sometimes so intolerable that the patients groan aloud, are covered with perspiration, and fall into convulsions.

As a rule, the pain is located immediately beneath the ensiform cartilage; in addition to diffuse tenderness, a circumscribed spot is especially painful. In other cases, the pain is situated under the sternum, in one of the hypochondriac spaces, lower part of the dorsal spine, or between the scapulae.

If the pain is especially severe in dorsal decubitus, the ulcer may be assumed to be situated on the posterior wall of the stomach, and the patients often assume a crouching position, bent over forwards. An ulcer on the anterior wall is associated with violent pain in abdominal decubitus. Left lateral decubitus is often assumed in ulcers of the pyloric portion, the opposite position in ulcers of the fundus and cardiac end.

It is not true that the occurrence of pain one or two hours after meals indicates that the ulcer is situated at the pylorus, nor that small ulcers cause slight pain.

The pain is sometimes produced by direct irritation of the ulcer by the ingesta (when it develops immediately after eating). In other cases the movements of the stomach affect the surface of the ulcer. If the pain is independent of the meals, it must be attributed to the further development of the ulcer and the injury of nerves in the walls of the stomach. Finally, severe pains may result from inflammation of the serous layer or adhesion of the stomach to adjacent organs.

Pains sometimes radiate into other nerve tracts (intercostal neuralgia and oppressed breathing, neuralgia of the left brachial plexus, pain in the shoulder).

Vomiting occurs with remarkable frequency, sometimes early in the morning or when the stomach is empty. The usually watery, mucous mass is generally alkaline and consists of saliva (it turns red on the addition of chloride of iron) which has been secreted in increased quantities and swallowed during the night. In other cases the ingestion of food produces vomiting, sometimes despite the utmost caution in diet. The food is vomited in an unchanged or slightly modified condition, and sometimes contains *sarcina ventriculi*. Bileous matter may also be vomited. The vomited matters will be tinged with blood if emesis constantly recurs and gives rise to small extravasations of blood upon the gastric mucous membrane, or if capillaries have been opened by the ulcerative process. In some cases the vomiting is so obstinate that the patients waste away to a skeleton.

Hæmatemesis is a very valuable sign from a diagnostic standpoint. It occurs spontaneously, after bodily or mental exertion, a heavy meal, or as the result of a blow on the epigastrium. It is apt to occur in women at the time of menstruation. Among 120 cases of round ulcer collected by L. Muller, hæmatemesis was observed in 35.

The patients occasionally state that something warm is trickling in the stomach, they have a peculiar taste of blood in the mouth, nausea occurs, and then bloody masses are vomited. If the hemorrhage is very profuse, the signs of sudden cerebral anemia develop (dizziness, syncope, tinnitus aurium, small pulse, etc.).

The amount of blood varies, but may reach several pounds. It is sometimes pure, sometimes mixed with food. It consists generally of dark, blackish-red, acid clots, which are not frothy. If a large arterial vessel has been opened, the blood is bright-red. The more profuse and sudden the hemorrhage, the earlier will vomiting occur and the less changed will be the blood. If the hemorrhage occurs gradually, the

coloring matter of the blood will be changed by the gastric juice and peculiar "coffee grounds" masses will make their appearance.

The hemorrhage is rarely the immediate cause of death. The patients generally recover gradually, and many suffer from repeated attacks.

If it is very profuse, it may enter the air-passages and be coughed up, so that patients who have suffered little from gastric symptoms may think that they have had an attack of hæmoptysis.

The appetite is sometimes unaffected, occasionally even increased, but many patients dread to satisfy it, because their discomforts are thereby increased. Thirst is often increased. Some patients suffer from profound depression, and occasionally from obstinate insomnia.

The general nutrition is sometimes so good that the patients present a blooming appearance. Rapid emaciation may occur when the nutrition suffers in consequence of the vomiting and violent pains.

The tongue is sometimes clear, sometimes coated. In many cases it has an extremely red, smooth or fissured surface, which has been attributed to partial desquamation of the epithelium from contact (during vomiting) with the acid contents of the stomach.

The epigastrium is occasionally distended. As a rule, it is tender on pressure, especially over a spot which corresponds to the situation of the ulcer. The lower dorsal or upper lumbar vertebræ are especially sensitive in ulceration of the lesser curvature and posterior wall of the stomach. Pain at the level of the umbilicus corresponds to ulceration of the greater curvature; pain in the right or left hypochondrium to ulceration of the pylorus or fundus.

If inanition threatens, the abdominal walls are sunken, the aorta is seen pulsating vigorously, and the spine and aorta are readily reached on palpation.

The bowels are usually constipated. Gastric hemorrhage is sometimes manifested solely by bloody, tarry, or black passages. But even when hæmatemesis is produced, the stools are almost always stained with blood.

The quantity of urine is generally diminished; the urine is concentrated and often very acid. Slight temporary albuminuria may develop after profuse hæmatemesis.

The duration of the disease is extremely variable. Brinton reports a case which lasted thirty-five years. Relapses occur very frequently, even at the end of years.

Rapid and permanent recovery is not very frequent. Even under favorable circumstances, a striking tenderness of the stomach often remains, and is manifested after every indiscretion in diet by a feeling of pressure, fullness in the epigastrium, severe gastric pains, and frequent emesis.

Among the sequelæ may be mentioned very violent gastric pains, which not infrequently continue for years, appear in paroxysms for a few days or weeks, and then disappear for a long time. In other cases, the symptoms of gastro-intestinal catarrh continue. If cicatrices have formed at the pylorus, stenosis of the pylorus and dilatation of the stomach (gastrectasia) will develop. Symptoms of pyloric insufficiency (incontinentia pylori) may be expected if the muscular coat has suffered serious injury.

In many cases in which the shape of the stomach has been materially changed by cicatrices, and constrictions of the cavity have formed, sudden symptoms of ileus may develop as soon as the communication be-

tween the anterior and posterior divisions of the stomach is interrupted.

If one of these divisions communicates with the colon by means of a fistula, lientery has been observed, *i. e.*, the food passes with great rapidity and very little changed into the colon, and very soon appears in the evacuations.

Hæmatemesis forms a transition between the symptoms proper and the complications. A very grave complication is cancer of the stomach, which develops not infrequently, at an advanced age, on the base of a round ulcer.

If signs of perforation appear, the situation becomes very grave. The patients usually complain of unspeakable pain, a feeling of annihilation, and sometimes of a sensation as if something had burst in the belly. The abdomen is tympanitic and extremely tender. Circumscribed and unusual areas of dulness, corresponding to exudation beneath the abdominal walls, make their appearance. If the liver and spleen are not bound down by old adhesions, the areas of dulness of these organs disappear, because the gas which escapes from the stomach separates these organs from the thoracic walls. Grave collapse is manifested by cool skin, small pulse, and sunken features. Vomiting is more frequently absent than present in perforation of the stomach. The sensorium is usually unclouded to the last moment. Death occurs with symptoms of increasing exhaustion or disturbances of respiration and circulation, as the diaphragm is pushed into the thoracic space to an extreme degree. Cases of recovery have been reported, but are extremely exceptional.

If the ulcer perforates through the anterior abdominal wall, cutaneous emphysema may develop from the passage of the gases of the stomach into the subcutaneous cellular tissue. The signs of pneumopericardium or pneumothorax (usually hydropneumopericardium or hydropneumothorax) rapidly develop after perforation into the pleural or pericardial cavities. Perforation into the lungs is recognized by the acid reaction of the sputum or the appearance of particles of food in it.

A gastric ulcer sometimes causes thrombosis of the portal vein and thus gives rise to the formation of numerous metastatic abscesses in various organs.

IV. DIAGNOSIS.—This is usually as easy in the majority of cases as it is impossible in rarer cases. The development of pain in the stomach, vomiting and hæmatemesis in a young, pale individual is almost always associated with a round ulcer of the stomach.

The diagnosis is difficult if it must be made from single symptoms. The disease may be mistaken for the following:

a. Chronic gastritis.—Errors of diet can usually be demonstrated as the cause; the tenderness is slighter but more diffuse; hæmatemesis is absent; a cure is more readily effected.

b. Pure nervous gastritis.—The pains are not closely associated with the meals, and are sometimes diminished by pressure in the epigastrium: hæmatemesis is absent; there are usually other nervous disturbances (intercostal neuralgia, clavus, etc.).

c. Cancer of the stomach.—The age of the patient is significant; cachexia soon develops; the left supraclavicular glands may be enlarged; the course of the disease is rapid; a tumor can be felt in the gastric region (cicatrices following ulcers are also perceptible to the touch).

d. Biliary colic.—The pains are confined chiefly to the region of the gall-bladder (outer border of the right rectus abdominis immediately be-

neath the right thorax); vomiting and chill often occur during the pains; scleral and even cutaneous jaundice develop not infrequently; the passages should be carefully examined for gall-stones.

V. PROGNOSIS.—A guarded prognosis should always be given. Even under favorable circumstances disturbances of digestion may persist for life, and on the other hand, hemorrhage or perforation may unexpectedly place the patient in great jeopardy. The prognosis is especially grave when we may assume that the ulcer is situated on the anterior wall of the stomach, since perforation is frequent in this locality.

VI. TREATMENT.—The patient should be kept quiet in bed until all acute inflammatory symptoms have subsided. A warm poultice should be applied constantly to the epigastrium. The chief importance should be attached to dietetic measures. If the ulcer is recent, solid food should be entirely avoided.

If the patient tolerates milk, this should form the sole article of diet for weeks, and may be taken boiled, warm or cold, as the patient desires. It should be taken often but in small quantities. If it is immaterial to the patient, the milk should be thoroughly boiled. If this gives rise to an excessive formation of acid in the stomach, sodium bicarbonate or lime-water should be added. When this proves ineffectual, a little flour may be added. I have recently obtained good results by mixing the milk with an equal quantity of soup. When milk produces a tendency to nausea or vomiting, Debove administers it through the œsophageal sound. When it is converted in the stomach into thick cheesy lumps, buttermilk should be given.

But if milk is not tolerated in any shape, we should order weak tea, meat soup, raw or soft boiled eggs (the latter taken in wine or bouillon, or seasoned with salt or sugar), and Leube-Rosenthal's meat solution. Rectal alimentation may also be resorted to.

Solid food may only be taken after acute symptoms have disappeared (vide Vol. II., page 63).

Among medicinal agents the Carlsbad waters enjoy the greatest repute. As a rule, the cooler wells (Schlossbrunnen, Marktbrunnen, Theresienbrunnen) are employed because the others are apt to produce gastric hemorrhage. If the treatment is to be carried out at home, one to three teaspoonfuls of Carlsbad salts dissolved in one-half litre water (50–55° C.) should be taken early in the morning (in three portions, one every ten minutes). Within two hours afterwards, the patient should have one or two thin, profuse evacuations. Breakfast may be taken half an hour after the last dose of the salts.

Acetate of lead (gr. $\frac{3}{4}$ every two hours) and nitrate of silver (gr. $\frac{1}{4}$ in pill t. i. d.) have been recommended to produce cicatrization, but much may not be expected from these agents.

If the pain is violent, we may order bismuth. subnitrat. gr. viij., morphin. hydrochloric. gr. $\frac{1}{2}$, ext. belladonna gr. $\frac{1}{4}$, sacch. alb. gr. v., one powder t. i. d. Subcutaneous injections of morphine in the epigastrium. chloral hydrate (gr. lxxv., tragacanthæ et pulv. althææ, q. s. u. f. pil. No. x. D. S. One pill t. i. d.), and warm compresses to the epigastrium may also be employed. Gerhardts extols liq. ferri sesquichlorat. (three or four drops in a wineglassful of water, several times a day).

In cases of obstinate vomiting, we may order the ingestion of small pieces of ice, subcutaneous injections of morphine, creasote (gtt. viij., aq. destil. $\frac{3}{4}$ iij. D. S. One tablespoonful every two hours), or tincture of iodine (gtt. viij., aq. destil. $\frac{3}{4}$ v. M. D. S. One tablespoonful every two hours).

Concerning the treatment of gastric hemorrhage, vid Vol. II., page 55. In recurring hemorrhages, Rydgier recommended the operative removal of the ulcer, and this was successfully performed by Kleef.

If signs of perforation-peritonitis develop, we may give large doses of opium, and cover the abdomen with light warm poultices.

When the acute symptoms have subsided, and anæmia is left over, preparations of iron may be cautiously administered.

APPENDIX.

Two other forms of ulceration occur upon the mucous membrane of the stomach, viz., the hemorrhagic erosion and the follicular ulcer.

a. The hemorrhagic erosions are very often found at autopsies. They consist of reddish, brownish, or blackish spots, which are sometimes round, sometimes elongated, and are situated at the top of the folds of the mucous membrane. They are sometimes so numerous that the mucous membrane presents a speckled appearance. They are found mainly or exclusively in the pyloric portion of the stomach.

Upon the application of a stream of water, a superficial loss of substance appears which rarely extends to the submucosa. The base is often villous, the edges slightly elevated and pulpy.

Hemorrhagic infarcts are found not infrequently in the immediate vicinity, and there are often gradual transitions between these, and more or less distinct losses of substance. In all probability, the hemorrhagic infarctions are converted into erosions, inasmuch as the extravasated blood interferes with the circulation, and thus exposes the affected part to the digestive action of the gastric juice. Hence gradual transitions are possible between erosion and round gastric ulcer.

Hemorrhagic erosions are more frequent in adults than in children. They result from all conditions which impede the circulation in the mucous membrane, or, in which the walls of the vessels have become abnormally permeable to red blood-globules. This includes emesis, gastric catarrh, ulcer and cancer, cough, pointed or irritating particles of food, disturbances in the portal circulation (diseases of the portal vein, liver, heart, and lungs), severe infections, febrile, exhausting diseases, scurvy, morbus maculosus Werlhofii, hæmophilia, etc. The lesion sometimes seems to develop during the agony, as the result of violent contractions of the stomach.

This condition is not recognizable during life. In rare cases, it may give rise to violent hemorrhage which may prove fatal.

b. Follicular ulcers are the result of inflammatory swelling and supuration of the lymphatic follicles. They may follow gastritis, but cannot be diagnosed during life.

7. *Cancer of the Stomach.*

I. ETIOLOGY.—Cancer of the stomach is rarer than round ulcer, for while the latter is found in five per cent of all autopsies, the former is found in only two per cent. Nothing is known positively concerning its etiology, except that it is almost always a disease of advanced age.

It is observed most frequently from the fortieth to the seventieth years of life, and is extremely rare before the age of thirty years.

Two cases of congenital cancer of the stomach have been observed, and one in a child æt. five weeks.

The male sex is said to be affected more frequently, but this statement has been disputed.

The poorer classes are affected somewhat more frequently than the well-to-do, probably because the excessive ingestion of vegetables by the former tasks the activity of the stomach, and often produces chronic gastric disorders.

There is no doubt that patients who suffer from chronic diseases of the stomach, particularly chronic gastritis and round ulcers, are predisposed in later years to cancer of the stomach. The disease has been associated, by many writers, with alcoholic excesses and injuries in the epigastrium. Pulmonary phthisis is also said to constitute a predisposing cause.

The statement that psychical causes exercise an etiological influence is entirely unfounded. According to some reports, climate exercises a certain influence. Thus, Griesinger observed no cases in Egypt, although gastro-intestinal diseases are very common. Heineman makes a similar statement concerning Vera Cruz. Autenrieth states that the disease is extremely frequent in Upper Suabia and the Black Forest, and attributes this fact to the excessive ingestion of farinaceous articles, potatoes, and sour articles of food. Cloquet makes a similar statement concerning Normandy, and explains it by the excessive drinking of cider.

II. ANATOMICAL CHANGES.—Cancer occurs more frequently in the stomach than in any other organ. In the large majority of cases, it is primary.

Secondary gastric cancer is remarkably rare; eight cases have been collected by Grawitz, four of which were secondary to œsophageal cancer, two to cancer of the rectum, one to cancer of the testicle, and one to cancer of the calf of the leg. Five additional cases have been since reported (primary site in the œsophagus, omentum, suprarenal capsules, and pelvis).

One of three varieties is usually observed: *a.* scirrhus; *b.* medullary or alveolar cancer; *c.* colloid cancer.

The most frequent form is scirrhus (this is firm, fibrous, and relatively dry); alveolar cancer is rarer (this is soft and juicy). The most infrequent form is colloid cancer. This is composed of a connective-tissue stroma forming cavities which are filled with yellowish or brownish gelatinous contents.

Scirrhus presents the most protracted course; alveolar cancer is characterized by a tendency to degeneration and the formation of metastases; and colloid cancer is apt to spread to the peritoneum.

Hard and soft cancer masses are observed not infrequently in the same case, and indeed a single cancerous nodule may present transitions from scirrhus to medullary cancer. Waldeyer applied the term carcinoma simplex to the transition stage between these two forms. If cylindrical cells predominate in medullary cancer, it is known as cylindrical epithelioma. Very vascular growths are called fungus hæmatodes.

Waldeyer teaches that cancer of the stomach is always of epithelial origin. It starts in a circumscribed proliferation of the principal cells of the tubular gastric glands, and these rupture through the muscular stratum of the mucosa. As soon as the proliferated cells reach the submucosa, they find a very suitable place for further growth, so that the submucosa often appears to be the starting-point of the tumor.

Cancer develops most frequently at the pylorus, not infrequently in the shape of a ring. Next in order of frequency are the lesser curvature and anterior wall of the stomach, then the posterior wall, cardia, and

greater curvature. It is observed only exceptionally at the fundus, even if almost the entire remainder of the organ is infiltrated.

Cancer of the cardiac end may spread to the œsophagus, that of the pylorus to the duodenum.

The cancer may form a circumscribed tumor or diffuse infiltration. In the former event, it may project into the cavity of the stomach like a fungus, sometimes with a smooth, sometimes with a villous surface (*carcinoma villosum*), which is often depressed in the centre (umbilication). The surface is not infrequently ulcerated, and the deep, crater-like loss of substance has a bloody, blackish, or discolored base. This should be attributed to the digestive action of the gastric juice. Circumscribed tumors are usually single, but multiple ones are not extremely rare.

Diffuse cancerous infiltration is most marked in the submucosa, and the latter is sometimes thickened three to five fold. The walls of the stomach are rigid, collapse very little or not at all, when the organ is opened, and present an unusual resistance to the knife. The surface of the mucous membrane is uneven and nodular, and is ulcerated whenever it is involved in the cancerous process. Ulceration and gangrene of cancerous tissue hardly ever occur in cancer of the stomach, probably because the gastric juice is strongly antiseptic.

The cancerous proliferation may extend to the muscular coat and even beneath the serous layer. In the former, it spreads particularly within the fibrous septa, the majority of which extend vertically through the muscular coat. The lymph channels also offer a favorable road for the growth, the endothelium of the lymphatics undergoing active proliferation.

The bundles of muscular fibres, which are surrounded by the cancerous septa, generally undergo hyperplasia, and their rosy color contrasts strongly with the white bands of cancerous tissue. Atrophic conditions of the muscular coat have also been observed in some cases.

From the connective-tissue strands of the muscular coat the cancerous new-formation extends to the subserous tissue, and there often spreads farther. The part played by the lymphatic vessels is recognized from the fact that the lymph-vessels of the serous layer are filled with firm cancer masses, thickened in places and dilated into the shape of a rosary. At times circumscribed fungoid tumors form which project above the serous layer into the peritoneal cavity. In those places at which the tumor touches neighboring organs, the abdominal walls or the diaphragm, are found similar tumors which depend upon local infection. Serious danger arises from the tendency of the cancer to spread and to ulcerate. The more it extends the more the stomach loses its digestive function and inanition threatens. Marked implication of the muscular coat of the stomach interferes with the movements of the organ which are so important in digestion. The progressive ulceration causes frequent hemorrhages, which are sometimes scanty and gradual, sometimes profuse. In addition, perforation may occur and be followed by peritonitis. Perforation may be prevented by preceding peritonitis and peritonitic adhesions to adjacent organs. But this only obviates the danger temporarily. The ulceration finally extends to the adherent organs, where it not infrequently gives rise to gangrenous changes. Abnormal communications may develop between the stomach and the colon, more rarely with the small intestine, anterior abdominal wall, pleural or pericardial cavities, lungs, and air passages. Hence manifold complications, such as pneumopericardium, hydropneumoperi-

dium, pneumothorax, hydropneumothorax, pneumonia, abscess and gangrene of the lungs. An external gastric fistula may be preceded by emphysema of the skin. In some cases, the ulceration extends to the spine, and finally causes changes in the spinal membranes, substance of the spinal cord, and nerves. In a few cases, the stage of ulceration is followed by the formation of a cicatrix. But the destruction generally proceeds further in other places, so that the occurrence of complete recovery by cicatricial formation has not been proven. Cicatrizing cancers with slightly prominent proliferations may be mistaken for granulating round ulcers. Vertical sections should then be made through the edges of the ulcers as far as the serous layer, and we should note whether cancerous proliferations can be detected in the muscular and subserous layers. Even the microscopical diagnosis may be difficult in firm cancers which are poor in cells. We should then look for cancerous degeneration of the epigastric glands. Hauser, who recently investigated the relations between gastric cancer and round ulcer, emphasizes the fact that at the edges of cicatrizing ulcers lively proliferation occurs into the stomach glands, and is apt to lead to cancer. Cancer not infrequently changes the shape and position of the organ. If situated at the pylorus, the stomach is often dilated, while it is exceedingly small if located at the cardiac extremity. Changes in shape are also produced by retraction of cancerous or cicatrizing masses. The weight of the tumor occasionally pulls the stomach downwards, so that the pylorus has been found in the iliac fossa, and even in the pelvis. The remainder of the mucous membrane of the stomach is often in a condition of chronic catarrh, and covered with ecchymoses or hemorrhagic erosions. Secondary cancer occurs not infrequently in other organs, sometimes from a direct spread of the growth, sometimes from metastasis. The latter may be produced by the agency of the lymphatic vessels and veins. Cancer elements which have been loosened from the mother tumor enter the circulatory channels, and are carried to other organs, where they remain and incite further proliferation. The retroperitoneal glands are usually in a condition of cancerous degeneration, thence the process spreads to the glands in the thorax and even to the peripheral glands (inguinal, supraclavicular). Cancerous growth has also been observed a number of times in the thoracic duct. Hepatic cancer or cancerous thrombosis of the portal vein occurs very often. The peritoneum, omentum, pancreas, spleen, kidney, pelvic organs, male sexual organs, pleura, lungs, brain or bones may also be affected. Among other organic changes, we may mention brown atrophy of the heart muscle, fatty degeneration of the heart and kidney, waxy degeneration, lymphoid medulla of the bones, and marantic thrombosis. Phthisical changes are often found in the lungs.

III. SYMPTOMS.—The most important symptom is the demonstration of gastric tumor. If we remember that in the healthy stomach the pylorus and lesser curvature are covered by the liver, and that the cardiac end is not in direct contact with the abdominal walls, it is evident that, as a general thing, gastric tumors will only become evident to the hand or eye when they have reached a very large size, or the stomach has been dislocated downwards so that the parts which are usually covered are in direct contact with the anterior abdominal walls. If a gastric tumor is evident to the eye, we generally find a round or elongated, occasionally nodular prominence, which does not move with the respiratory movements. We must avoid mistaking the respiratory sliding of

the abdominal walls over the tumor with a respiratory dislocation of the tumor itself. The growth sometimes changes its position with the position of the body. It is generally situated so much higher the more the stomach is filled with food, and it may even disappear beneath the liver. Prominences of less size are better seen on oblique illumination of the abdominal walls. The tumor is found most frequently in the epigastrium, the larger portion being situated to the right of the median line; it is also seen in the umbilical region and lower down, and more rarely in the hypochondrium. On palpation we not infrequently recognize a tumor which is not visible to the eye. Sometimes we must remain satisfied with the demonstration of increased resistance in the region of the stomach. The tumor is often uneven, nodular, and sensitive on pressure. It may sometimes be moved to a certain extent in the abdominal cavity. Sometimes it can be felt only at times and disappears, particularly when the stomach is full. A tumor may sometimes be reached more readily in the knee-elbow position. It has often been found to increase in size during protracted observation, or sometimes to grow smaller. To demonstrate the connection of the tumor with the stomach, the latter may be distended with carbonic acid gas, according to Frerich's method. A teaspoonful of tartaric acid in a little water is given to the patient and then an equal amount of sodium bicarbonate. As the position of the stomach changes with its distention, the tumor will be more or less distinctly dislocated. If the patient is very greatly emaciated, pulsation of the tumor will be seen occasionally and felt even more frequently. The pulsations are conveyed from the aorta and are never diffusely pulsating in character.

Respiratory displacement of a gastric tumor will occur when the latter is adherent to the liver or spleen, and the dislocation is conveyed from these organs, or when the tumor is very large, so that the walls of the stomach are diffusely infiltrated with cancer, and therefore cannot yield to the respiratory compression produced by the diaphragm.

As a rule, percussion shows a dull tympanitic note over the tumor and the tympanitic quality is rarely absent. Auscultation of the stomach gives no decisive results; but if the aorta is compressed, a systolic murmur of stenosis may be heard. The diagnosis is generally more or less doubtful if a tumor cannot be demonstrated. Hemorrhage from the stomach is practically important in diagnosis. The vomiting of dark, clotted blood is more rare than in gastric ulcer. In the majority of cases smaller hemorrhages occur, the blood remains for some time in the stomach and is changed by the gastric juice, and coffee grounds, chocolate, or inky masses are vomited. Under the microscope these are found to consist of constituents of the food and discolored, more or less changed red blood-globules. (Vide Vol. II., fig. 9.) This form of vomiting occurs not only in cancer of the stomach; it also takes place, though much more rarely, in gastric ulcers.

In suspicious cases, the stools should be carefully examined, since vomiting may be absent in slight hemorrhages, and the blood passes through the intestines. In other cases, the hemorrhage is so profuse that death is the immediate result. Vomiting of mucoid, biliary matter or of articles of food occurs very often, and is especially significant of cancer when advanced age, increasing emaciation, cachexia, and cancerous degeneration of peripheral glands, especially the left clavicular glands, are associated with one another. According to Bamberger, *sarcina ventriculi* occurs more frequently in the vomited matter than in other diseases of the stomach. Obstinate vomiting sometimes ceases suddenly. This

occurs in pyloric cancer when the increasing destruction of the tumor renders free an existing narrowing, or when the stomach is diffusely degenerated and no longer produces vigorous muscular contractions. Sometimes this is a symptom of exhaustion. Riegel, who found that free hydrochloric acid was very generally absent in the gastric juice in cancer, recently observed that the cancer proliferation can give rise to rapid disappearance of hydrochloric acid when present. Wolff and Quetsch agree that the absorption of potassium iodide is delayed. Methyl violet is the most practical test of the presence of free hydrochloric acid in the gastric juice or contents. If to a solution of 0.05 : 200 a fluid is added which contains minimum amounts of hydrochloric acid, the violet color is changed to a dark blue at the point of contact of the fluids. Other free acids give a similar, though not so decided reaction, so that a negative result alone proves the absence of hydrochloric acid. To study the absorption, 0.2 of potassium iodide in a gelatin capsule should be given to the patient and the saliva examined for iodine every five to ten minutes. A piece of starch paper moistened with saliva is touched with a trace of pure nitric acid, and the presence of iodine is shown by the yellowish-red color of the paper. The statements with regard to the normal rapidity of absorption differ very widely.

Among the other symptoms of cancer, the signs of marasmus and disturbed digestion are the most important. The patients often attract our attention by rapid emaciation, a sallow or greenish complexion, loss of strength, and œdema of the ankles. The skin is usually lean, thin, extremely dry, desquamating, and itching. Indeed, violent pruritus in cachectic individuals points to latent cancer, if albumin and sugar are not present in the urine. If local changes are absent, the history may be very similar to that of progressive pernicious anæmia, especially as the blood is often very pale, almost serous, and poor in red blood-globules. The latter may be irregular and unequal in size (poikilocytosis). The majority of patients complain of loss of appetite, rarely the desire for food is unchanged, or boulimia is present (especially in stenosing cancers of the cardiac portion). Increased thirst has been observed a number of times. The weight of the body diminishes constantly, but in rare exceptions a temporary increase of weight is observed. Elevation of bodily temperature occurs at times, probably as the result of septic fever from absorption of degenerating masses of cancer. The patients often suffer from obstinate insomnia. Many are tortured by boring, burning pain in the stomach. It is almost constant, increases after meals, and sometimes becomes excessive at night. The pain may radiate into adjacent nerve tracts and give rise to asthmatic and stenocardiac disturbances. The epigastrium is sensitive on pressure, sometimes over a localized spot corresponding to the tumor. As a general thing, however, the pains are not so violent as in round ulcers.

The tongue sometimes has a grayish-white or brownish coating, sometimes is very red, clean, fissured, the latter especially when very sour masses are vomited. Increased secretion of saliva has also been observed. Ebstein noticed spasm of the pharynx, which interfered with deglutition and appeared to be reflex. Frerichs noticed eructation of inflammable gases.

At first the bowels are almost always constipated. Later obstinate diarrhœa sometimes occurs and has been attributed to intestinal catarrh resulting from decomposition of the food by ulcerated portions of the cancer. Dysenteric symptoms (tenesmus, bloody and purulent stools) are observed occasionally. The urine is generally scanty, high-colored, and occasionally contains a very large amount of indican. The color becomes a reddish-blue or deep blue, if a test tube is half filled with urine, the other half with pure hydrochloric acid, and one to three drops of a fresh concentrated solution of chloride of lime are cautiously added. Jaksch

observed acetone in a few cases (Burgundy color of the urine on the addition of a very dilute solution of chloride of iron). Maixner found peptone in the urine in a number of cases.

Special symptoms develop not infrequently when the cancer narrows the pyloric or cardiac orifices. In the former event, the signs of dilatation of the stomach gradually develop. But if the cancer destroys the pyloric muscle in places and thus makes it incapable of function, the symptoms of incontinence of the pylorus may be produced. Cancer of the cardiac orifice occasionally gives rise to symptoms of stenosis of the œsophagus. The patients cannot swallow the food, and after a while it regurgitates in a macerated condition. On auscultation of the œsophagus, the murmur of deglutition diminishes or ceases at the level of the eleventh dorsal vertebra. The introduction of the œsophageal sound meets with an obstruction at the same locality, and particles of cancer occasionally remain in the fenestra of the sound. (Vide Vol. II., Fig. 7.) The patients emaciate with extreme rapidity, and generally complain of a feeling of hunger. The duration of gastric cancer is determined with difficulty, because the duration of the latent stage is unknown. In some cases death is said to have occurred at the end of the first month, in others not until the third year. A year may be regarded as the average duration. In many cases death follows with the signs of increasing marasmus. The patients waste away to a skeleton, and become more apathetic with each succeeding day. (Edema of the subcutaneous tissue and serous cavities often occurs and finally death ensues. Slight albuminuria or marantic thrombosis of a lower extremity is observed in some cases. Inanition delirium may precede death. In some cases death is the result of intercurrent diseases, such as pneumonia. Sometimes it occurs unexpectedly as the result of complications, for example, profuse hemorrhage, perforation-peritonitis, or rupture into other organs; but fistulæ may be tolerated for a long time. This includes stomach-colon fistula, the chief characteristics of which have been considered in the description of the round gastric ulcer. Williams reports a case in which perforation of the stomach, occurring upon sitting up, was accompanied by a slight noise. Sometimes the symptoms of secondary cancer in other organs, especially the liver, predominate to such an extent that the primary gastric cancer is overlooked.)

IV. DIAGNOSIS.—The diagnosis is not easy, and we often find nothing during life, although the autopsy reveals a cancerous tumor of considerable size in the stomach. If a tumor is visible or palpable during life, it should be remembered, in differentiating it from tumors of the liver and spleen, that it does not move on respiration, apart from the exceptions mentioned on page 79. We must also avoid mistaking it for coprostasis, intestinal tumors, tumors of the omentum and pancreas, diseases of the lymphatic glands, tumors of the uterus and ovaries, aneurisms of the aorta and celiac axis, or encapsulated peritonitic exudations.

Apart from other symptoms, we must lay special stress upon the fact that gastric tumors change their position more or less with the distention of the stomach. But not every tumor of the stomach is cancerous, and hypertrophy of the muscular coat, cicatrices following round ulcer, or foreign bodies may be mistaken for it. Advanced age and very marked cachexia testify in favor of cancer, and these factors are also important in the differential diagnosis from ulcer, cardialgia, and chronic gastritis. Cancerous tumors of the lymphatic glands above the left

clavicle, although they are by no means constantly present, are very important in diagnosis. But these should not be mistaken for tubercular glands, and it must be remembered that the swelling affects only the glands on the left side, that is, those which are adjacent to the point of entrance of the thoracic duct.

These cancerous glands are also very important from a diagnostic standpoint, in cases of general marasmus whose origin we must investigate. The demonstration of dilatation of the stomach or incontinence of the pylorus may also be important, provided that other causes for these conditions may be excluded. The condition is suspicious when the patients suffer from pruritus of the skin and when free hydrochloric acid cannot be found in the gastric juice or contents, and when absorption can be shown to be slower than normal.

V. PROGNOSIS.—The prognosis is bad. Permanent recovery is hardly possible, even when the tumor is removed by operation.

VI. TREATMENT.—The use of condurango is said to have produced recovery. Although this drug will not cure cancer, it is, nevertheless, an excellent stomachic which often increases the appetite, stops the vomiting, and diminishes the pain. The following prescription may be given.

R Corticis condurango,	5 ss.
Macera horas xii. c. aq.,	3 xij.
Dein coq. ad remanent col.,	5 vi.
D. S. One tablespoonful two to three times a day.	

In addition we should order light, nutritious food, according to the principles laid down on page 63, improve digestion by the administration of hydrochloric acid and, if stenosis of the cardiac extremity is present, maintain life by nutritive enemata (vide Vol. II., page 33). In addition, symptomatic treatment must be adopted (narcotics against pain, etc.). Billroth has recently resected the cancerous stomach. Among seven of his cases three died, while Czerny had two deaths in four operations. The patient to be operated on should still be in possession of a certain amount of vigor, the cancerous degeneration should not be too extensive and, if possible, adhesion to adjacent organs and metastases should be absent. The success of the operation depends in great part upon the period at which it is performed. According to Czerny, thirty-six operations had been performed up to the beginning of 1884, among which twenty-seven ended fatally; eighteen of these died within the first twenty-four hours after the operation, so that this was probably performed too late, but it is probable that recovery will be temporary even in the most favorable cases.

APPENDIX.

The other neoplasms of the stomach hardly possess more than an anatomical interest, not that they are destitute of symptoms, but on the demonstration of a tumor, we will always be led to diagnose a gastric cancer. We may make brief mention of the following tumors:

a. Gastric polypi are usually the result of chronic gastritis. They may be single or multiple, of very unequal size, and are sometimes the product of a proliferation of the mucous membrane, sometimes of that of the submucous tissue. In one case Cruveilhier observed stenosis of the pylorus by a polypus.

6. Sarcoma. Myosarcoma has been found a number of times, and in one case smooth muscular fibres were found in the secondary sarcoma nodules of the liver.

Papilloma, myoma, adenoma, cysts, teleangiectesia, lymphangioma, lipoma, and gumma, have also been described.

1. Dilatation of the Stomach.

(Gastroectasia. Dilatatio ventriculi.)

I. ETIOLOGY. The conditions necessary to dilatation of the stomach are always furnished when the forces necessary to propel the gastric contents are insufficient. Such conditions may develop either because unusual obstacles are situated at the pylorus or because the muscular coat of the stomach is paralyzed, or finally because the amount of ingesta is unusually large. As a matter of course, some of these factors have only a temporary effect, so that we may differentiate an acute and chronic dilatation of the stomach. For example, an unusually heavy meal may produce an acute dilatation. Chronic conditions of gastric dilatation have a special clinical interest, and the following remarks will refer chiefly to them. Stenosis at the pylorus is the most frequent and important of all the causes. It may affect either the pylorus directly, or the first part of the duodenum.

The most frequent lesion is a cicatrix at the pyloric ring, usually the result of a preceding ulcer, more rarely of poisoning by caustics, finally of cancerous degeneration. But benign hypertrophy of the pylorus, such as occurs in chronic gastritis, or when polypi have entered the pylorus, may also give rise to stenosis. In some cases cicatrices or tumors are situated in the first part of the small intestine, but produce the same effect as pure stenosis of the pylorus. Tumors of the adjacent organs may also compress and narrow the pylorus or duodenum.

Bartels and Mueller-Warneck first called attention to the association of dilatation of the stomach with floating kidney (right side). Two cases of this kind have recently come under my observation.

Landerer recently called attention to the not infrequent occurrence of congenital stenosis of the pylorus which is followed by dilatation of the stomach. Sometimes twists occur at the pylorus or duodenum and are followed by gastroectasia. In one case Kussmaul observed that on the filling of the stomach the cancerous pylorus, with the remainder of the stomach, turned around the long axis of the organ, at the same time pushed from before backwards, against the entrance to the small intestine and occluded it like a valve. In other cases the twist occurs between the first horizontal and vertical sections of the duodenum.

Dilatation of the stomach also occurs not infrequently from disease of the muscular coat, either as the result of local or general causes. Among the local causes is chronic gastritis; the same effect is also produced by destruction of the muscular coat by extensive ulcers or cancerous infiltration, especially if the points of insertion of the circular muscular bundles are destroyed. Traube showed that in many cases of ulcer the gastric branches of the vagus are destroyed, thus giving rise to a diminution in the tonus of the organ. The muscular power of the stomach is occasionally interfered with by peritonitic adhesions to the anterior abdominal walls or adjacent organs, or because the organ is pulled upon by the transverse colon which has entered a hernia. Gastric dilatation has also been observed as the result of injury in the region of the stomach. Among the general causes which may weaken the muscular coat of the stomach we may mention chlorosis, anæmia, phthisis, typhoid fever, pyæmia, diseases of the brain and spinal cord, hysteria, and hypochondria (atonic gastroectasia.)

It should also be mentioned that the stomach sometimes assumes a vertical position, either as a congenital condition or as the result of tight corseting or of tumors in the abdominal cavity. This condition also predisposes to gastric dilatation, especially of the pyloric portion, because the propulsion of the gastric contents is impeded. It may also develop when the abdominal walls are very flaccid and the recti abdominis are separated from one another.

Finally dilatation of the stomach develops when an excessive demand is made upon its energy. It is remarkably frequent in heavy feeders, and also in countrymen who live on a chiefly vegetable diet. It is often observed in diabetics and in them is attributed to the excessive ingestion of food and drink. The ingestion of indigestible articles also gives rise to gastroectasia.

Neuwerk reports a case in which in a girl, *æt.* 23 years, the signs of acute stenosis of the pylorus developed after eating cherries. Dilatation of the stomach occurred later, and at the autopsy, three months afterwards, ten cherry pits were found in the stomach.

Dilatation of the stomach is not a very rare disease. It is not so infrequent even in childhood. According to Comby it is relatively common in rachitic children. It develops most frequently from the fortieth to the fiftieth years, and is more frequent in males than in females.

II. ANATOMICAL CHANGES.—Dilatation of the stomach may attain a remarkable size. Cases are known in which, upon opening the abdomen, very little but the stomach was visible and the greater curvature extended into the pelvis. In Jadon's case the cavity of the organ is said to have contained ninety pounds of fluid. The spleen and liver are usually found pushed upwards, the small intestines downwards and to the sides, the heart is also pushed upwards; atrophic changes are occasionally noticed in the displaced organs. If the dilatation is in the first stages of development, it is found, as a rule, to be most marked at the fundus. Circumscribed and diverticulum-like sacculations of the stomach are observed very rarely, and are generally produced by foreign bodies or peritoneal adhesions. If the dilatation is the result of pyloric or duodenal stenosis, the œsophagus sometimes takes part in the dilatation. The mucous membrane of the dilated organ is generally in a condition of chronic catarrh. The muscular coat is sometimes thickened three or four fold, sometimes thin and atrophic, sometimes thickened and thinned in places. As a matter of course, the condition of the muscular coat depends upon the causes of the disease. Thickening, to a certain extent of a compensatory character, is to be expected in stenosis of the pylorus, while atrophic conditions are apt to develop in direct affections of the muscular coat, especially in conditions of general weakness. Fatty and colloid degeneration of the muscular fibres are often, though not constantly, found on microscopical examination.

III. SYMPTOMS.—Two groups of symptoms must be sharply differentiated from each other, *viz.*, the chemical and physical changes. The former are the result of the stasis of the gastric contents and of abnormal fermentation, and even gangrenous decomposition, while the physical changes depend entirely on the increased size of the organ. As a rule, medical aid is sought on account of the chemical changes.

In the beginning, the disturbances of gastric digestion are so slight that the condition is apt to be regarded as catarrh of the stomach. As the disease progresses, disturbances of nutrition are not long delayed. The patients emaciate, the complexion grows pale, the skin thin and dry,

the muscles become soft, and sometimes the bones appear under the skin like those of a skeleton. The face usually has an ashen and wrinkled appearance.

The appetite is diminished in many cases, in others boulimia is present. The latter is especially true when the dilatation is the result of marked stenosis of the pylorus, so that very little food passes into the duodenum, while the chief mass of food remains in the stomach and is vomited from time to time. Furthermore, Fader and Penzoldt have shown that absorption is rendered slower in the dilated stomach. Thirst is often increased because fluid is absorbed with difficulty, and very often little passes into the intestines. Many patients complain of eructations, heart-burn, and pyrosis. Sometimes odorless gases are eructated, sometimes they have the foul odor of sulphuretted hydrogen, even a gangrenous odor has been noticed. Eructation of inflammable gases has been described in a number of cases. In Frerichs' case the patient noticed that, upon eructating while lighting a cigar, the gas caught fire so that his moustache was burned. In a case reported by Friedreich, in which the flame was occasionally one-third meter in length, marsh gas was found in the eructated gases. The chief constituents are oxygen and nitrogen in the same proportions as in atmospheric air (probably swallowed air), hydrogen and carbonic acid (from fermentation of carbohydrates), in a few cases marsh gas, and in one case an oil-producing gas.

The following are the results of the chemical analysis of the gases:

	Frerichs.	Popoff.	Schultze.
Carbonic acid,	20.57 per cent.	12.82 per cent.	26.56 per cent.
Hydrogen,	20.57 "	32.32 "	32.30 "
Marsh gas,	10.75 "		0.34 "
Oil-producing gas,	0.20 "		
Oxygen,	6.52 "	7.9 "	0.36 "
Nitrogen,	41.38 "	46.5 "	33.44 "

Vomiting is an almost constant symptom. It is especially early and profuse when the disease is caused by stenosis of the pylorus. If the stomach gradually increases in size, the vomiting again becomes more infrequent and often occurs only at intervals, perhaps every three or four days. Astonishing amounts are sometimes vomited, in some cases as much as sixteen pounds at one time.

The vomited matters almost always have a strongly acid reaction. Many patients complain that the teeth feel extremely blunt after the vomiting, and a rapidly progressing affection of the teeth is often noticeable. The smell of the vomited matters is often intensely sour; in other cases, rancid and sweetish. In rare cases the odor is gangrenous. This occurs, it appears, with relative frequency in carcinoma. The reaction should always be tested with litmus paper, because a sour smell is not positive evidence of an acid reaction. The vomited matter is sometimes fluid, sometimes like porridge, depending chiefly upon the character of the food. This is also true of its color. If the dilatation is due to cancerous degeneration, the vomited matters may be colored like chocolate, coffee grounds, or ink. It usually separates very quickly into three layers; the uppermost consisting chiefly of frothy masses, the middle one of fluid, and the lowermost of a crumbly sediment. If kept for a few hours, it often continues to ferment.

Lactic, butyric, and acetic acids have been repeatedly demonstrated in

the vomited matter; peptone, undigested albumen, starch, and sugar have also been found.

On microscopical examination, we find more or less changed débris of food, *sarcina ventriculi*, yeast cells, and schizomycetes (vide Fig. 10). In two cases Nannyn found mould fungi.

Vomiting often occurs with extreme facility. As the disease approaches a fatal termination, it may cease completely. A very noteworthy feature is the unusually long stay of food in the stomach. In washing out the healthy stomach, in a fasting condition early in the day, the fluid flows out clear, while the dilated stomach always contains more or less considerable remains of the food taken on the preceding day. Faver and Penzoldt showed that absorption in the stomach is delayed in gastroæctasia.

FIG. 10.



Sarcina ventriculi (sc) from the vomited matters in gastric dilatation following a stenosing pyloric cicatrix after round ulcer. On the right a few yeast cells (hf). At the edges three swollen vegetable cells from the food. Enlarged 275 diameters.

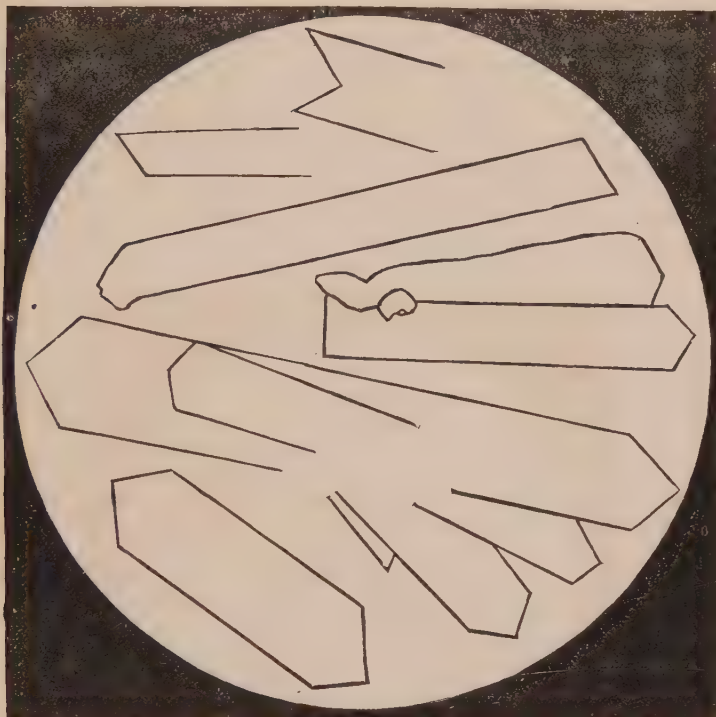
Frequent and profuse vomiting exercises a great effect upon the stools and the urine. The bowels are usually constipated, the patient often going from one to two weeks without an evacuation.

The urine is often alkaline. This reaction is explained by the fact that the blood becomes poor in acids on account of the profuse and very acid vomiting.

Ebstein, Stein, and Scherf showed that the alkaline reaction of the urine results in the formation of unusual sediments (triple phosphates and crystals of phosphate of magnesia, vide Fig. 11.) If a twenty-per-cent solution of ammonium carbonate is added to the latter, the crystals undergo disintegration at once. If triple phosphates or phosphate of lime are also present in the sediment, the reagent produces no change in the former, while the phosphate of lime is gradually destroyed after a long time.

The profuse vomiting also diminishes the amount of urine, and many cases have been observed in which the daily amount fell to 400-300

FIG. 11.



Crystals of phosphate of magnesia from the alkaline urine in dilatation of the stomach. After Ebstein and Scherf.

ccm. As a matter of course, its chemical constitution changes. Scherf obtained the following results in two cases :

	Daily amount in ccm.	Specific grav- ity.	Daily amount of urea in gms.	Daily amount of chlorides in gms.	Daily amount of phosphoric acid in gms.
CASE 1.					
Average.....	1030	1022	14.8	8.11	1.19
Maximum.....	2220	1027	22.7	14.3	1.69
Minimum.....	500	1013	8.5	3.0	0.75
CASE 2.					
Average.....	1350	1018	22.7	11.9	1.16
Maximum.....	2200	1025	35.0	16.6	1.48
Minimum.....	600	1012	17.1	8.5	0.54

There are not a few subjective symptoms in this disease. The patients are particularly annoyed by emesis, heartburn, a feeling of fulness

and pressure in the stomach, and increasing loss of strength. Attacks of dyspnoea and palpitation may occur if the development of gas in the stomach is very considerable and interferes with the mobility of the diaphragm.

But these symptoms merely result from abnormal fermentation of the gastric contents, and the demonstration of dilatation of the stomach can only be determined by physical examination.

On inspection, we notice not infrequently an unusual prominence of the gastric region. In advanced cases, this extends below the umbilicus. The greater curvature of the stomach can often be followed with the eye, and occasionally the lesser curvature is also visible at a little distance below the ensiform cartilage. As a matter of course, the entire organ is situated abnormally low in such cases, since under normal conditions the lesser curvature lies behind the liver.

The peristaltic movements of the stomach may be very active, particularly when the gastric dilatation is the result of pyloric stenosis. The waves of contraction generally run from the cardiac to the pyloric extremities, rarely in the opposite direction. Bamberger reported a case in which a deep constriction formed at about the middle of the stomach, whence the peristaltic contractions travelled in both directions. These movements may often be provoked by pinching or faradization of the abdominal walls, or douching with cold water. The occurrence of these contractions below the umbilicus is especially important in the diagnosis of gastroectasia, because the greater curvature of the healthy stomach is generally situated above the navel.

Kussmaul states that unusually active peristalsis of the stomach may give rise to a disagreeable feeling of movement in the abdomen (peristaltic restlessness of the stomach). The waves of contraction require about a minute to travel from the cardiac to the pyloric extremity. They are particularly apt to occur in cicatricial stenosis of the pylorus or first part of the duodenum.

Palpation reveals a peculiar feeling of resistance which is recognized on short, quick strokes of palpation. A sensation is felt as if we were touching a distended air bag. If palpation, or rather immediate percussion, is carried out from above downwards, we can often recognize the lower border of the stomach with great distinctness. If the peculiar feeling of resistance extends below the umbilicus, the diagnosis of gastric dilatation can usually be made with certainty.

If the stomach contains gas and fluid, as is generally the case, vigorous shaking movements will give rise to very loud splashing sounds. These are sometimes heard throughout the entire room.

These sounds may also be heard in the healthy stomach when the latter contains gas and fluid. In gastroectasia, however, the sounds are unusually frequent and loud.

The splashing sounds can sometimes be felt as fluctuation. If the patient sits up, the fluid in the stomach will accumulate in the greater curvature, and the lower border of the organ can sometimes be determined by the feeling of fluctuation. If fluctuation is recognizable below the umbilicus, the diagnosis of dilatation of the stomach may be made.

Examination with the œsophageal sound may furnish extremely important results (vide p. 30). Leube showed that the sound, when introduced into the dead body, may push that portion of the wall of the stomach which is situated opposite the cardiac extremity to a line connecting the anterior superior spinous processes of the ilea. In individuals

with thin abdominal walls, the tip of the sound may be felt from the outside during life. Indeed, in combined examination from the abdominal walls and rectum, the sound may sometimes be felt between the examining fingers. Leube concludes, therefore, that dilatation of the stomach may be diagnosed if the tip of the sound can be felt below the above-mentioned horizontal line. But this mode of examination is not devoid of danger, particularly if recent ulcers are situated on the gastric mucous membrane.

Penzoldt employs another method of examination. He found that in the healthy individual the sound may be introduced about 60 centimetres before meeting with the resistance of the wall of the stomach which is situated opposite the cardiac extremity. In three cases of gastric dilatation this distance amounted to 70 centimetres.

Purgecz fastened a manometer to the posterior extremity of the œsophageal sound. So long as the sound remained in the œsophagus, the manometer showed negative pressure, but this became positive as soon as it entered the cavity of the stomach. From this point Purgecz could advance the sound (in healthy individuals) from 27 to 30 centimetres before meeting with the resistance of the lower border of the stomach. In gastroectasia this distance was considerably greater.

Oser calls attention to the fact that in both these methods of examination the sound may perhaps glide along the greater curvature to the pylorus, where it first causes a feeling of resistance, *i. e.*, a stomach appears to be dilated, although in reality it possesses normal dimensions.

The symptoms obtained on percussion are extremely important in diagnosis. Percussion of the dilated stomach almost always gives a deep, tympanitic sound, which not infrequently possesses a metallic quality. If the stomach contains fluid, a dull percussion sound will be heard over the corresponding region, and this will vary with the position of the body. If the stomach contains but little fluid, it may accumulate, in dorsal decubitus, along the posterior wall, while in the vertical position it is situated along the greater curvature. Hence the entire gastric region will give a tympanitic sound during dorsal decubitus, but in the erect position a strip of dulness makes its appearance, corresponding to the greater curvature. Gastroectasia is present if this strip of dulness is situated below the umbilicus.

If dulness has been detected in the region of the greater curvature, and the fluid contained in the organ is then removed by means of the stomach-pump, the previously dull region will become tympanitic.

It has been shown that if one litre of fluid is drunk in a fasting condition, a strip of dulness as broad as a finger will make its appearance along the greater curvature of the stomach, while the patient is in an erect position. In healthy individuals this area of dulness is situated above the umbilicus.

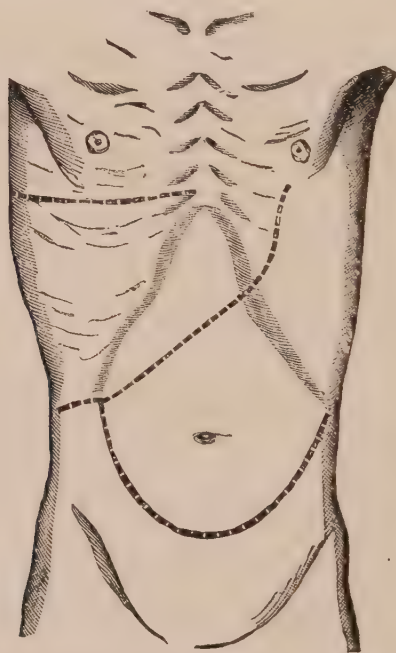
Frerichs' method, which we described above, is the most rapid and certain for percussing the distended stomach. This makes the contours of the stomach more distinct, so that they are appreciable to the hand, the eye, and the percussion hammer with great facility. Mannkopf and Wagner showed that the lower curvature of the healthy stomach never lies beneath the umbilicus, and dilatation of the stomach is thus readily recognized. (Vide Fig. 12.) In percussion of the stomach when distended with carbonic acid, we should not be satisfied with determining a single point of the lower border of the organ, because dilatation can only be diagnosed if, in addition to abnormal depression of the lower

border, the lateral boundaries of the percussion figure of the stomach extend at least to the normal borders, that is, on the left side to the anterior axillary line, on the right to the parasternal line. If the lateral borders approach closer to one another, the depression of the lower border of the stomach must be attributed to congenital or acquired vertical position of the non-dilated organ.

Distention of the stomach sometimes remains absent after the development of carbonic acid gas, and acute intestinal tympanites develops in its stead. This is an evidence of incontinence of the pylorus.

Recently, in employing this method, I have not been satisfied with percussion alone, but have also resorted to auscultation. Auscultation of the stomach shows an extremely fine crackling, which results from a development of fine bubbles of carbonic acid. This ceases abruptly at

FIG. 12.



The percussion boundary of the dilated stomach (which has been distended with carbonic acid), in a woman *æt.* 37 years.

the boundary of the greater curvature of the stomach. Bamberger auscultated the stomach during drinking. This produces the impression as if a drop of water is dropping into a large empty cavity. It is said that this falling of drops cannot be heard beyond the greater curvature.

In some cases a crackling murmur in the region of the stomach can be heard without previous artificial distention with carbonic acid, but in my experience the former is coarser than the murmur described above, and is owing to the development of gas produced by fermentation of the gastric contents.

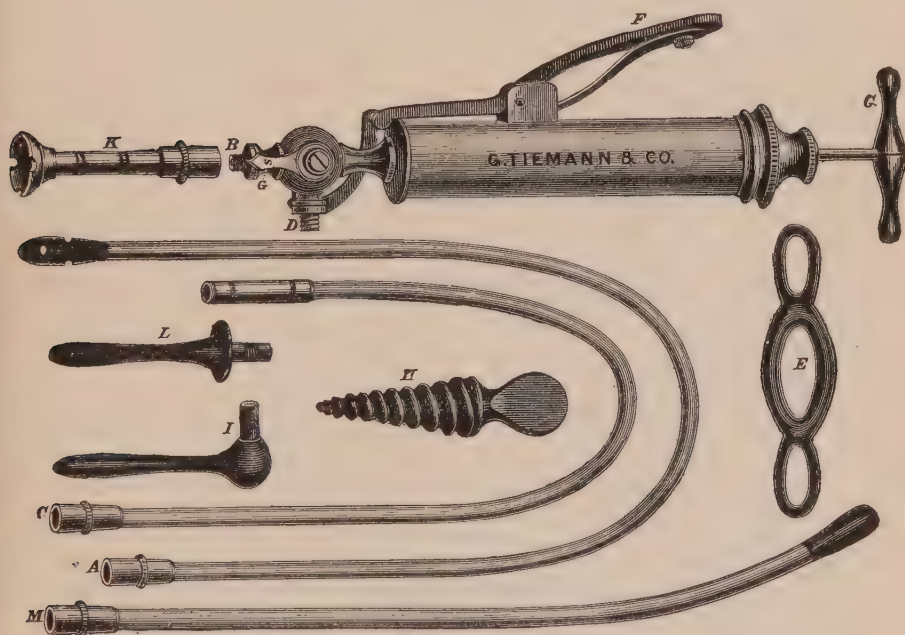
Sometimes it is difficult to demonstrate gastroectasia despite all the methods mentioned. It is difficult, particularly, to distinguish the lower

border of the stomach from the transverse colon. Under such circumstances we may inject fluid or air into the transverse colon through the rectum. The injection of water is best effected by means of a funnel and rubber tube, through which water is allowed to flow into the intestine.

To inject air we may employ a flexible tube, which is connected with the balloon of the Richardson ether spray apparatus. Naunyn recently emphasizes the fact that microscopical examination of the vomited matter or of the contents of the stomach which have been removed by the stomach-pump is important in diagnosis, because yeast fungus and schizomycetes indicate abnormal fermentation of the gastric contents, and this occurs with greatest frequency in gastroæctasia.

The dilated stomach may displace adjacent organs. The apex beat

FIG. 13.



Stomach pump.

of the heart is often found in the fourth intercostal space. The movements of the heart are sometimes extremely distinct, because the organ is pressed forcibly against the anterior chest-walls. Metallic resonance of the heart-sounds in the dilated cavity of the stomach is observed occasionally, and this may increase the intensity of the sounds to such an extent that they may be heard at some distance from the patient. The more the heart is pushed upwards the larger Traube's semilunar space becomes.

The liver and spleen are also often pushed upwards.

The disease is almost always chronic. If it is incurable on account of the primary affection, the strength of the patient diminishes more and more, œdema develops, and the patient finally dies with symptoms of inanition and marasmus. Bamberger observed a sort of spontaneous

recovery in pyloric stenosis, inasmuch as unchanged portions of the pylorus yielded and thus eliminated the constricting action of the cicatricial tissue.

IV. DIAGNOSIS.—The recognition of dilatation of the stomach will generally present no special difficulties when the methods of examination described above are properly employed. But the diagnosis is only complete when the cause of the dilatation is recognized. For this purpose the clinical history must be relied upon.

V. PROGNOSIS.—This is unfavorable in many cases, because we are unable to relieve the primary affection. This is especially true of many cases which are the result of pyloric stenosis, but the tendency to relapse is also marked under other conditions, and we must therefore be very cautious in giving a favorable prognosis.

VI. TREATMENT.—The object of local treatment is to relieve the stomach of its excessive and decomposing contents, and thus to prevent further fermentation of the food. The removal of the gastric contents is best effected by means of a stomach pump or stomach siphon.

The stomach pump (vide Fig. 13) is an aspiration syringe, which possesses two outlets at its anterior extremity.

By means of a valvular arrangement at the side of the syringe, it is possible, when the valve is untouched, to have only one opening in communication with the interior of the syringe, so that when this is connected with a sound which is introduced into the stomach, and the piston of the syringe is withdrawn, the contents of the stomach will be sucked into the interior of the syringe. After the syringe is filled, the valvular apparatus is pressed downwards. The opening which connects with the stomach-tube is thus closed, while the other, previously closed opening now communicates with the cavity of the syringe. If the piston is now driven home, the contents of the syringe can be expelled. This procedure may be repeated until the stomach is empty.

Force must be avoided, since otherwise pieces of the mucous membrane of the stomach may be aspirated and torn off. This accident hitherto has been unattended with any special danger.

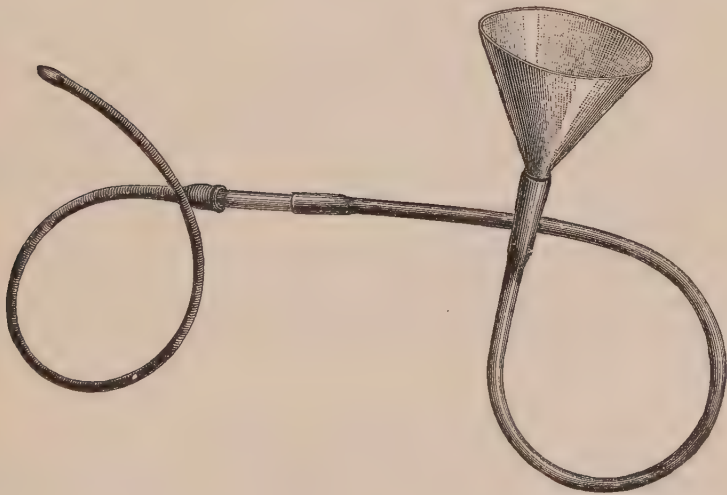
This danger is lessened by the employment of the stomach siphon. The following is the simplest arrangement of this apparatus:

A rubber tube, one metre in length, is fastened to an œsophageal sound (this should be made of soft rubber) by means of a glass tube, about fifteen cm. in length (vide Fig. 14). The free end of the tube is now provided with a glass funnel. The sound is introduced into the stomach and fluid is poured into the funnel. Before all of the fluid has escaped from the funnel, the tube is firmly pressed between the thumb and index finger, and the funnel is bent downwards and placed in a vessel. The fluid which has been poured in and the contents of the stomach will now escape spontaneously. If the fenestra of the sound is choked up by undissolved fragments of food, the patient should be allowed cautiously to cough or to bear down, or the region of the stomach may be compressed. Sudden and repeated compression of the rubber tube is often sufficient to remove the obstacle. If this does not prove successful, water should again be poured into the funnel, and the entire procedure be repeated. When the contents of the stomach have the consistence of a thick porridge, it is best to dilute them by large amounts of water. But we should carefully measure the quantity removed in order to prevent overloading of the stomach. The first manipulations with the instrument are apt to be attended with difficulty, because they produce nausea and

perhaps attacks of dyspnœa and palpitation of the part. The operation is best performed in the morning in a fasting condition, the water being at a temperature of 30°. After the stomach is empty, it should be washed with a solution of Carlsbad salts (one teaspoonful of the salt to one litre of water at 30° R.), until the outflowing water is alkaline. We may then inject to advantage anti-fermentative remedies, such as resorcin (one per cent), natrium salicylicum (one per cent), creasote, benzol, carbolic acid, permanganate of potash. The patient should take no food for several hours after the operation. It should be repeated daily, and the intervals should be increased only after the symptoms of fermentation of the food have disappeared. Double sounds have been employed by some writers, but in our opinion they are unnecessary. Some physicians make use of an ordinary rubber tube.

As a rule, the patients feel marked relief after artificial emptying of the stomach. In others, dizziness, ringing in the ears, twitching in in-

FIG. 14.



Stomach siphon of soft rubber.

dividual muscles, and even extensive muscular spasms have been produced; but these symptoms usually possess no serious significance. One case, however, has been reported in which the patient employed this method himself in an irrational manner, and died in an attack of tetany.

After the stomach is emptied, the patient is allowed to rest for several hours, during which an ice-bag is applied to the region of the stomach. I have often obtained very good results from subcutaneous injections of ergotinum Bombelon or strychnine (gr. iss. : 3 iij., 2 to 3 minims), faradization of the stomach—measures which should be employed immediately after the stomach is empty. The patient should wear a tight abdominal bandage while walking about.

The faradic current should be employed by placing one electrode on the left hypochondrium, while the other is slowly passed over the region

of the stomach from the cardiac to the pyloric extremity; strong current, moist electrodes; duration of the sitting, five minutes.

Scanty meals should be frequently taken, perhaps every two hours. Fluids, carbohydrates, and fats should be avoided as much as possible, the diet consisting chiefly of animal food (vide Vol. II., page 63). Milk, and milk mixed with pieces of ice, are especially indicated when the vomiting is very violent, but small quantities only should be taken at one time.

Digestion may be improved by giving a few drops of hydrochloric acid (five drops to a wineglassful of lukewarm water) half an hour after the principal meal. Two or three hours later we may give resorcin (gr. v.) or carbolic acid (gr. xxx., pulv. althææ, q. s. ut f. pil. No. 25. D. S. 1 pill t. i. d.). As a rule, favorable effects soon follow the local treatment of dilatation of the stomach; the patients become more cheerful, their appearance improves, and the weight of the body increases. Vomiting, pyrosis, and eructations disappear; the urine becomes more abundant and acid; evacuations from the bowels are more frequent, and the boundaries of the stomach approach the normal.

General treatment must also be adopted. We must pay especial attention to anæmic and nervous conditions (preparations of iron, nervines, cold-water treatment, sea-bathing, etc.).

As a rule, treatment must be continued for a long time. Indeed, if stenosis of the pylorus is present, it must be continued for life. The patients soon learn to introduce the stomach tube, and to empty and wash out the stomach without the aid of the physician. This may be done two or three times a week, but should be intrusted only to intelligent patients.

Resection of the pylorus (pylorectomy) has been performed a number of times with success. Loreta attempted dilatation of the pylorus in two men suffering from cicatricial stenosis, the stomach being laid free, and both index fingers being introduced into the pylorus through the external wall of the stomach.

9. *Incontinence of the Pylorus.*

1. This condition is recognized by the fact that the stomach can be distended only temporarily or not at all by Frerichs' method. In its stead we notice acute tympanites of the intestines, because the gas rapidly passes through the open pylorus. The entire abdominal walls become distended within a few minutes, and the ascending and descending colon are especially apt to project as prominences which are as thick as a man's arm.

2. It is produced by interference with the muscular layer of the pyloric ring, either of an anatomical or purely functional character. The former may be produced by round ulcer, or cancerous infiltration and ulceration. The latter is generally produced by cerebral or peripheral disturbances of innervation. The latter category probably includes attacks of acute tympanites in hysterical individuals. Ebstein also found incontinence of the pylorus in a woman suffering from softening of the spinal cord, while the walls of the stomach were anatomically intact. I have repeatedly observed transitory incontinence of the pylorus. It occurred in individuals suffering from acute or chronic gastritis, with violent exacerbations, and the incontinence disappeared as soon as the gastric affection was relieved. Perhaps it resulted from paralysis of the

muscular fibres of the pylorus in consequence of inflammatory, serous infiltration. According to Kussmaul, the signs of incontinence of the pylorus are presented by healthy individuals who are in a fasting condition.

3. Incontinence of the pylorus may produce injurious effects. The very acid contents of the stomach may give rise to diarrhœa by the unusual irritation of the intestinal mucous membrane. On the other hand, the occurrence of the incontinence may improve or entirely relieve previous vomiting. Stenosis and incontinence of the pylorus may exist side by side. If the pyloric ring is converted by cancer into a narrow rigid fissure, which is incapable of contraction, the conditions are furnished both for stenosis and incontinence.

4. The treatment must be directed against the primary affection.

10. *Degenerative Changes in the Stomach.*

Degenerative changes in the walls of the stomach hardly possess more than an anatomical interest; fatty degeneration (especially after phosphorus poisoning and anæmic conditions), calcification (in carious and other processes associated with the destruction of bone) and waxy degeneration have been described. Occlusion of the blood-vessels by waxy degeneration may cause ulceration of the mucous membrane.

In several cases of waxy degeneration, Elinger was unable to detect free hydrochloric acid in the gastric juice. It is therefore possible to recognize this change, if, under conditions in which waxy degeneration is apt to develop, free hydrochloric acid cannot be found in the gastric juice, provided cancer may be excluded. Elinger obtained the gastric juice by allowing the patient to swallow small sponges which were fastened to a thread and which after a time were withdrawn from the stomach and compressed. If free hydrochloric acid is present, a yellow solution of tropæolin (gr. $\frac{2}{4}$: $\frac{3}{4}$ vij.) turns red, and methyl aniline violet (gr. viiss. : $\frac{3}{4}$ vij.) dark blue.

11. *Atrophic Changes in the Stomach.*

Fenwick has recently shown that disappearance of the glands may produce very dangerous conditions. In some cases this disease is independent. In others it is associated with other gastric affections or follows cancerous disease in other organs, and marantic conditions in general.

The peptic glands disappear in places and are replaced by connective tissue; some of them have become cystic. The process seems to be the result of an interstitial interglandular proliferation of connective tissue. In other respects the condition of the stomach depends upon the primary disease; sometimes it is small and thin, sometimes dilated or thickened.

A few cases run their course with the symptoms of progressive pernicious anæmia. The condition cannot be diagnosed with certainty.

12. *Softening of the Stomach. (Gastromalacia.)*

1. Softening of the stomach is most frequent in children until the end of the second year, especially after artificial feeding and diseases of the stomach. In adults it has been observed in sudden death after a

heavy meal, or after diseases of the brain, of the spinal cord, typhoid fever, pyæmia, dysentery, etc.

2. The condition is easily recognized anatomically. In the milder grades, the mucous membrane is found softened in places, swollen, easily removed with a knife, and sometimes even by a stream of water. In advanced cases the softening extends to the muscular coat, so that the stomach is only held together by thin serous membrane. The latter often ruptures on contact, and the contents of the stomach pass into the peritoneal cavity. In many cases the rupture of the stomach occurs before the abdominal cavity is opened, especially if the corpse has not been carried with great care. The lower third of the œsophagus may also show softening, and if this organ is perforated, the contents of the stomach enter the pleural cavity. The adjacent portions of the diaphragm may also be softened and destroyed. Although other organs may be affected, the softening is always most marked in the stomach. The color of the softened tissue is not always the same, so that we are accustomed to speak of white, brown, and black softening. In white softening, the swollen tissue is gray or milky white, and in a few places we recognize brownish streaks and lines which correspond to the larger distended blood-vessels. The other coats of the organ are usually very anæmic. In brown or black softening, we have to deal with a brownish or blackish pulpy mass, the color of which is dependent on the distention of the veins of the mucous membrane.

Occasionally the softened tissue may assume some other color corresponding to that of the food taken just before death. Under the microscope, the epithelium and connective tissue are found more or less dissolved and destroyed, while the smooth muscular fibres and elastic tissue are intact, and can thus be readily separated from one another.

3. Elsaesser stoutly maintains that all these changes are post-mortem. His principal reasons for holding this position were the following: *a.* the absence of all symptoms of inflammation, even after perforation of the stomach; *b.* the influence of age and diet. The softening is especially frequent in children, because in them the tissue possesses very little resistance, and the contents of the stomach have a special tendency to acid fermentation. *c.* The softening only extends as far as the contents of the stomach are in contact with its walls. *d.* It can be produced in other portions of the stomach, in addition to the fundus, by changing the position of the body. *e.* It is found especially in the summer months, because the stomach then undergoes cooling very slowly, and the gastric juice exercises its solvent action for a long time.

In recent times, however, a number of cases have again been reported in which brown softening is said to have occurred at the close of life. The question cannot, however, be settled with positiveness. At all events, the lesions belong more to pathological anatomy than to clinical observation.

13. *Rupture of the Stomach. (Gastrorhexis.)*

1. Rupture of the stomach may occur from without inwards or in the opposite direction. Traumatic rupture is infrequent. Buyst has recently described a case in which a fall, without external injury of the abdominal wall, produced rupture of the pylorus, duodenum, and spleen. Perforations sometimes occur into the stomach from adjacent organs, for example, in abscesses of the liver, spleen, or peritoneum, purulent

pleurisy and pericarditis, tuberculosis of the spine and ribs, etc. Diseases of the walls of the stomach, especially ulcer, cancer, and toxic gastritis, are a frequent cause of perforation. It is occasionally produced by the ingestion of sharp, hard, or insoluble foreign bodies. It has also been maintained that an excessively heavy meal or abundant development of gas may produce spontaneous rupture of the stomach, despite the fact that the walls of the organ are healthy. The reported cases are not reliable.

2. Sudden pain, signs of grave collapse, extensive tympanites, and symptoms of perforation-peritonitis (pain, disappearance of hepatic and splenic dulness) are mentioned as the chief symptoms of rupture of the stomach. Vomiting may be absent. A rupture is sometimes said to be attended with a loud noise. Emphysema of the skin developed in a case reported by Newman, probably as the result of injury of the parietal peritoneum.

3. The prognosis is bad. The treatment consists of the administration of opium and stimulants.

14. *Animal and Vegetable Parasites in the Stomach.*

1. Among the animal parasites may be mentioned *trichina spiralis*, *ascaris lumbricoides*, and tape worms. These not infrequently produce intolerable gastralgia, which ceases abruptly when the worms are vomited. It may also be mentioned that *ecchinococci* may enter the stomach from the liver, spleen, omentum. Meschede observed a very violent gastritis in a boy after eating cheese. This disappeared after living cheese mites were vomited. Gerhardt also observed acute gastritis, which disappeared after the vomiting of larvæ of diptera. The diagnosis can only be made during life after the worms have been vomited. Caution must be exercised, particularly in hysterical females who sometimes bring living frogs, snails, etc., to the physician, with the claim that these had been ejected from the stomach.

2. The vegetable parasites include *sarcina ventriculi*, yeast fungus, sprue, mould fungus, and bacteria.

Sarcina ventriculi is often found. Its peculiar shape (vide Figs. 9 and 10) renders a mistake hardly possible. It may occur in the gastric contents of healthy individuals. Bamberger believes that its abundant development is characteristic of gastric cancer. Its mode of development is unknown. Its power of resistance is very great; for example, Duckworth kept it for three years in closed tubes. Yeast fungus is very intimately connected with fermentative processes in the gastric contents, but scattered yeast cells are found very often in vomited matters without justifying us in assuming abnormal fermentation. Their shape is easily recognized (vide Vol. II., Fig. 10). *Oidium albicans* (sprue) does not occur often in the stomach. Its appearance is shown in Fig. 2. Masses of sprue sometimes spread directly from the œsophagus to the mucous membrane of the stomach. In other cases the sprue develops in the stomach, although the œsophagus is intact. In a woman who died of cholera, Rudenew found *penicillium glaucum*, the fungus infiltrating two small tumors situated near the pylorus. Naunyn also observed mould fungus, in two cases, in the gastric contents. *Schizomycetes* are always found in the contents of the stomach. Their relation to the digestive processes still remains to be explained. They increase very markedly in number in abnormal fermentation of the gastric contents. Klebs re-

cently described brown spots upon the mucous membrane of the stomach, which consisted of colonies of bacteria, situated partly free in the lumen of the glands, partly between the epithelium and the membrana propria. In addition there was an inflammatory accumulation of round cells between the glands. He called the bacterium *bacillus polystorus brevis s. gastricus* (5.9–11.56 μ in length and 11.47 μ in width.)

15. *Foreign Bodies in the Stomach.*

Foreign bodies are rarely swallowed with suicidal intent; they are sometimes found in the stomach of the insane; thus Baillarger reported a case in which a lunatic swallowed a fork 14 cm. in length. Six years later, at the autopsy, the fork (blackened and covered in places with a reddish coating) was found in the stomach. Mountebanks sometimes suffer from this accident. Gussenbauer recently reported a case in which a sword swallower, who had introduced the sword into the stomach, broke the weapon by an incautious movement of the head, and a piece 20 cm. in length and 2 cm. in width entered the stomach.

Russell and others have described cases in which women had acquired the habit of swallowing the hairs which remained in the comb after their toilet. Gradually the stomach became filled with a large mass of hair which could be felt during life, and was regarded as a tumor. A case of this kind recently came under my own observation, but in this instance the hair was discharged through the rectum.

Foreign bodies, even when they possess very large dimensions, may be tolerated by the stomach for a long time.

In some cases they produce the symptoms of pyloric stenosis or inflammation of the walls of the stomach which may even lead to perforation. On the other hand, the foreign body may pass through the stomach and give rise to disease of the intestines, for example, typhlitis. A large proportion of the foreign bodies pass off with the stool without giving rise to any symptoms. This is true even of pointed objects such as needles.

Expectant measures must be adopted. Porridge, mashed potatoes, and similar articles of food may be given, and, if necessary, gastrotomy must be performed.

16. *Changes in the Shape and Position of the Stomach.*

1. Changes in the shape of the stomach are congenital or acquired. Stokes recently described a deep constriction at the middle of the stomach, which produced no symptoms during life. We have previously referred to the constrictions which are occasionally produced by ulcerative and cicatrizing processes in the wall of the organ. These changes can sometimes be recognized during life, by the employment of Frerichs' method. Buhl has also described an excellent example of congenital occlusion of the pylorus.

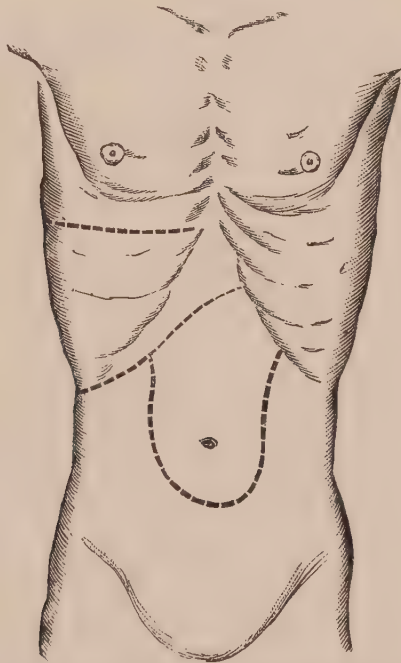
2. Among the changes of position we may mention transposition of the viscera. This generally affects all the other abdominal and thoracic organs. The stomach is situated on the right side, the liver on the left, the cardiac extremity is directed towards the right, the pylorus toward the left. This condition is generally recognized with facility, particularly if the stomach is dilated with carbonic acid, because Traube's semi-

lunar space will be present to the right in the hepatic region instead of a dull percussion sound.

Not very infrequently we notice an abnormal vertical position of the stomach so that the pylorus is situated lower and is nearer to the median line. This condition may be congenital or acquired. The latter particularly in women who wear tight stays, and in large tumors of the liver or spleen. This is easily recognized on distention of the stomach with carbonic acid. The lower border of the stomach is beneath the umbilicus, but the percussion figure of the organ is smaller from the right to the left, and extends very little or not at all beyond the median line of the abdomen. (Vide Figs. 15 and 12.)

We have previously mentioned that a vertical position of the stomach predisposes to gastroectasia, and abnormally low position of the stom-

FIG. 15.



Outline of the stomach on percussion in vertical position of the organ.

ach is found when tumors of the organ have dragged it downward. Peritoneal adhesions may also give rise to an unusual position of the organ. The stomach is sometimes found to be abnormally high, so that the upper border of the semilunar space extends to the fourth left intercostal space, and also possesses unusual dimensions in the left side of the thorax. In cases of diaphragmatic hernia the stomach may be situated in the left pleural cavity.

APPENDIX.

Changes in the gastric vessels are not very infrequent, but they rarely constitute an independent affection. Ponfick described aneurism of the

right gastro-epiploic artery in a woman who suffered from repeated attacks of pain around the gall-bladder and epigastrium, and suddenly died from rupture of the aneurism. Aneurism of the right coronary artery of the stomach has also been observed. *b.* Embolism and thrombosis of the gastric arteries play an important part, as we have previously mentioned, in the development of the round gastric ulcer. Infectious emboli may produce abscess of the stomach.

B. FUNCTIONAL DISEASES OF THE STOMACH. NEUROSES OF THE STOMACH.

1. *Rumination (Merycismus).*

I. ETIOLOGY. Rumination in man is a rare phenomenon. Bourneville and Séglas were able to collate only forty-six cases. It must not be forgotten, however, that many patients conceal the disease.

It is more frequent in men than in women, and almost always before the age of twenty years. It sometimes seems to be inherited by the children of the patients, but this apparent heredity may perhaps be explained as the result of imitation, which undoubtedly plays a great part in the etiology of rumination. In some cases the disease seems to be the result of congenital weakness of the muscular coat of the stomach. These individuals are generally able to force food from the stomach into the buccal cavity by slight pressure upon the epigastrium, deep inspirations, laughing or vigorous muscular exertion. This perhaps occurs spontaneously at first, is continued out of sport, and finally becomes pronounced rumination.

The condition has sometimes been attributed to errors of diet (imperfect mastication, excess of vegetables, etc.). Occasionally it results from gastro-intestinal diseases (chronic gastritis, obstinate constipation, a blow upon the stomach). Cold and mental excitement have also been mentioned among the causes.

A predisposition to the disease is furnished by anæmia, hypochondria, hysteria, epilepsy, idiocy, insanity, chorea, onanism. In one case it was preceded by an attack of whooping-cough.

II. SYMPTOMS.—Some time after eating, the patients regurgitate the food into the mouth (at first voluntarily, then involuntarily), and then expectorate it, swallow it forthwith, or masticate it again and then swallow it. Rumination may begin five to ten minutes after the meal and last five to six hours or more. Some patients ruminate after each meal, others only after they have eaten rapidly or have partaken excessively of vegetables, or after obstinate constipation, etc. Rumination may be repressed by some patients, in others this is no longer possible.

In many cases the general condition is unaffected. In others, gradual emaciation occurs, and finally a dangerous condition of exhaustion, particularly when the ruminated food is at once expectorated. Other patients present symptoms of gastric catarrh, especially if the regurgitated food is immediately swallowed in an imperfectly masticated condition, and irritates the gastric mucous membrane. According to Canstatt, emesis is produced with difficulty in these patients.

The disease often lasts for life. In others it ceases abruptly after marriage or after recovery from a previous gastro-intestinal affection.

III. ANATOMICAL CHANGES.—A dilatation (the so-called præstomach) is sometimes observed at the transition of the œsophagus into the stomach, sometimes above, sometimes below the diaphragm. But this condition is sometimes accidental, sometimes secondary to the rumination.

IV. DIAGNOSIS, PROGNOSIS, TREATMENT.—The absence of œsophageal disease or over-distention of the stomach distinguishes rumination from the regurgitation observed in such conditions. The disease is rarely attended with serious danger, and recovery generally follows if the co-operation of the patient is secured.

The diet should be carefully regulated. Fluid food in small quantities should be given every two hours, a daily evacuation from the bowels should be secured, and any primary affection which may be present should receive suitable treatment. At the same time the patients should endeavor to repress the act of rumination by all the power of their will. Koerner has recently recommended very highly the ingestion of pieces of ice immediately after meals. Perhaps the cold stimulates the cardia to more vigorous contractions.

2. *Peristaltic Restlessness of the Stomach.*

(*Tormenta ventriculi nervosa.*)

1. Under the term peristaltic restlessness of the stomach, Kussmaul has described conditions of unusually vigorous movements of the stomach. The muscular contractions are visible under the abdominal walls, moving slowly from right to left, the walls of the stomach occasionally rise very high in places. In addition, there is a feeling of restlessness and moving to and fro in the gastric region. The most varied splashing noises are often heard on auscultation of the stomach. The intestines may also take part in the peristaltic restlessness, although constipation is sometimes present because the large intestine is unaffected. These phenomena appear particularly after meals, but may also occur while fasting, or even at night.

2. Such conditions may be the result of mechanical causes, and occur in stenosis of the pylorus (vide page 88), but they also occur as an independent neurosis, and are produced by mental excitement, sexual excesses, anæmia, and general nervousness. Stiller regards them as the result of spastic closure of the pylorus.

3. Treatment consists in careful regulation of the diet and stools. In addition, rest in bed, warm poultices, and treatment of the primary disease.

3. *Hypersecretion of the Gastric Mucous Membrane.*

Reichmänn describes a case of idiopathic hypersecretion of the gastric mucous membrane in a man æt. 27 years, who was annoyed during the night by spasm of the stomach, heart-burn, and increased thirst. In addition, increased appetite, no signs of an anatomically demonstrable disease of the stomach, and constipation. If the stomach-pump was used early in the morning, after fasting for some time, 180–300 ccm. of a fluid, which was colored green with bile, could be removed from the stomach. This had an acid reaction, and digested fibrin completely in seven minutes at 40° C. It contained no diastatic ferment, and therefore did not consist of swallowed saliva (vide p. 60). The patient was cured by restriction of the amount of fluids, meat diet, washing out of the stomach morning and evening, poultices to the abdomen, and enemata to relieve thirst.

4. *Nervous Pain in the Stomach. Gastralgia.*

(*Cardialgia. Gastrodynia.*)

I. ETIOLOGY.—Pain in the stomach is sometimes produced by anatomical changes, sometimes it occurs as an independent nervous affection. The latter alone will now be considered.

This is found not infrequently in chlorosis and conditions of convalescence and exhaustion.

Gastralgia seems to be a neuralgia of the sensory nerves of the stomach, perhaps as the result of perverse processes of nutrition. It occurs not infrequently in onanists and those who indulge in sexual and alcoholic excesses. It is also very frequent in phthisis and Bright's disease, even in the early stages.

Certain general diseases may also give rise to attacks of spasm of the

stomach. Thus gastralgia may take the place of or precede an attack of gout. It develops occasionally as the result of malaria, occurring in periodical attacks which are only relieved by quinine or arsenic.

In some cases the disease is the result of an affection of the brain, spinal cord, or peripheral nerves. Attacks may occur during the course of locomotor ataxia (*Crises gastriques*). They have also been observed in softening of the brain and tumors of the pneumogastric and sympathetic. Furthermore, they occur not infrequently in hysteria, neurasthenia, and hypochondria.

Sometimes we have to deal with a reflex disease, provoked by affections of other abdominal organs. This is observed most frequently in women who suffer from diseases of the uterus or ovaries. The gastralgia occurs occasionally at the period of menstruation. It may also develop in the course of diseases of the bladder, kidneys, liver, pancreas, spleen, and intestines.

In many cases no cause is demonstrable.

Gastralgia is much more frequent in women than in men, and occurs generally from the fifteenth to the fortieth years. It is rare in childhood, more frequent in old age.

II. SYMPTOMS.—The chief symptom of the disease is pain in the stomach, which begins suddenly or is preceded by prodromata (feeling of fulness in the stomach, frequent eructations, nausea, vomiting, mental depression, pain in the head, etc.).

The pain may be intolerable. It is described as boring, burning, sticking, spasmodic, and is located chiefly in the epigastric region. But it often extends into the back between the scapulae into the umbilical region and the hypochondrium.

It is often increased by gentle pressure, while it is usually relieved by more vigorous pressure. Hence many patients firmly press the hands into the epigastrium, or assume the abdominal position, or are bent over forwards.

The epigastrium is often sunken. The abdominal walls are hard and contracted, and we can often recognize the pulsation of the aorta. In other cases the epigastrium is distended, and the stomach itself tense and prominent.

The attacks of pain often occur without an exciting cause. In other cases they are preceded by mental or bodily exertion. They appear very frequently in the fasting condition, and are often relieved by eating. Many patients complain of *boulimia*, or desire unusual, often very indigestible articles of food.

The duration of the pain is extremely variable. Sometimes it lasts a few minutes, sometimes several hours. Only a single attack may occur, or they may be repeated daily and even several times a day for weeks, months, or years. The gastralgia of intermittent fever occurs at definite intervals at certain hours of the day. In women gastralgia sometimes occurs only at the period of menstruation, or it is produced by certain manipulations upon the sexual organs. (In Niemeyer's case it occurred whenever leeches were applied to the *os uteri*.) At the height of a paroxysm many patients are terrified by a feeling of annihilation; the face is pale, the skin cool; the pulse is small and irregular, sometimes slow, sometimes rapid; perspiration breaks out over the body. In some cases we notice syncope, and partial muscular spasms or general convulsions.

The end of the attack is often announced by special symptoms, such as eructations, vomiting, yawning, etc. Constipation is generally present

at the period of the attacks. The urine is often scanty, and shows an abundant brick-red sediment. In hysterical patients, on the other hand, a very clear and pale urine (*urina spastica*) is often discharged at the close of the attack.

Fischl has observed albuminuria which sometimes lasted for several days. Hyaline casts also were found in the urine. The albuminuria is said to be owing to the fact that the pain diminishes the pressure of blood in the renal artery.

Gastralgia sometimes alternates with other neuralgias. In rarer cases it is present at the same time.

III. DIAGNOSIS.—The recognition of gastralgia is not always easy, and we must differentiate it from the following diseases:

(a) *Rheumatism of the abdominal muscles.* In this disease the pain is less paroxysmal; it is increased on pressure, and not infrequently jumps from one place to another.

(b) *Neuralgia of the lower intercostal nerves.* The pain can be traced along a single intercostal space; Valleix's painful points can generally be demonstrated; gastric symptoms are absent.

(c) *Circumscribed peritonitis.* There is great tenderness on gentle pressure, and true paroxysms of pain are absent; the etiology should also be taken into consideration.

(d) *Biliary colic.* The pain is generally confined to the region of the gall-bladder (external border of the right rectus muscle, immediately below the edge of the thorax); signs of jaundice; passage of gall-stones in the stools.

(e) *Intestinal colic.* The pains change their situation more rapidly, and signs of accumulation of gas in the intestines are noticeable.

(f) *Radiated pains.* Pains in the epigastrium occur in renal colic, pleurisy, and pericarditis, but careful examination of the organs will establish the diagnosis.

In addition we should endeavor to determine from the clinical history whether the gastralgia is a purely nervous affection or the result of anatomical changes in the walls of the stomach.

IV. PROGNOSIS.—This is favorable, in so far as death from gastralgia is unknown. Recovery will not occur unless the cause can be removed.

In some cases the disease continues for years.

V. TREATMENT.—The first indication is the treatment of the primary affection. To combat the gastralgia itself, we may apply a warm poultice to the region of the stomach, after a subcutaneous injection of morphine has been made. If syncope occur, the patient should inhale ammonia or eau de cologne, and five drops of ether or twenty drops tincture of valerian may be given internally. An immense number of other remedies have been recommended, of which we may mention: narcotics (opium, chloral hydrate, belladonna, chloroform, etc.), nervines (bismuth, nitrate of silver, Fowler's solution), sinapisms, irritating inunctions.

Kussmaul and Marlfranc report very good results from washing out the stomach. Two to three litres of a carbonated fluid at 38° are injected into the stomach every morning by means of the ordinary siphon apparatus, and then removed.

Electricity is often serviceable. Indeed Leube maintains that, when it is ineffective, the gastralgia is the result of anatomical changes in the stomach.

Leube applied the anode of the constant current to the painful part

in the region of the stomach, the cathode to the axillary region, or to the spine. (Strong stable current of five to ten minutes' duration.) Others recommend the faradic current, either both poles to the abdominal walls, or one electrode is passed into the stomach through a rubber tube, the other is applied externally.

5. *Nervous Dyspepsia.*

(*Neurasthenia Gastrica.*)

I. SYMPTOMS.—Dyspepsia implies difficult digestion, especially in the stomach. It may accompany various gastric affections, or appear as an independent neurosis. The latter will alone be considered.

In nervous dyspepsia the mechanical and chemical digestion in the stomach does not appear to be changed, but nevertheless the digestive process is attended with local disturbances and general nervous symptoms. The latter must be regarded as reflex.

The symptoms occur after a heavy meal, generally after dinner, and gradually subside toward the end of digestion, *i. e.*, in five or six hours. The patients have a disagreeable sensation of fulness and pressure in the region of the stomach despite careful regulation of the diet. Sometimes there is decided pain which is usually lessened by pressure on the stomach. Eructation is a frequent symptom. The patients complain of heartburn, sometimes of nausea and vomiting. There is usually anorexia, more rarely boulimia. Thirst is occasionally increased, the tongue is not coated, sometimes a bad or perverse taste in the mouth is noticed. Some patients have a feeling of constriction along the œsophagus; the bowels are usually constipated.

General nervous symptoms are very common. The patients complain of a rush of blood to the head, dulness, ringing in the ears, spots before the eyes, pain in the head, and dizziness. They are depressed and moody. Sleep is disturbed, but sometimes there is marked drowsiness and constant yawning after a hearty meal. Asthmatic attacks or palpitation of the heart may be prominent symptoms. The patients grow pale and gradually emaciate.

II. ETIOLOGY.—The disease is more frequent in men than in women, and begins generally between the ages of thirty and forty years. It often accompanies other neuroses, for example, hysteria, hypochondria, neurasthenia, and insanity. In other cases it is associated with anæmia and conditions of weakness (chlorosis, phthisis, Bright's disease, syphilis, lactation, etc.). It sometimes seems to be the result of toxic causes (excessive use of alcohol or tobacco, uræmia, and malaria).

Nervous dyspepsia is sometimes a sequel of other diseases of the stomach, for example, chronic gastritis and round ulcer. Finally, it may develop in a reflex way in diseases of the intestines, uterus, ovaries, and kidneys.

III.—DIAGNOSIS AND PROGNOSIS.—The diagnosis cannot be made with certainty unless we are able to exclude anatomical diseases. Leube attaches great importance to the fact that the chemical digestion in the stomach is properly performed. The prognosis should be given with caution, because complete and permanent recovery is not frequent.

IV. TREATMENT.—The improvement of the general condition is much more important than regulation of the diet. We may resort to sea baths, cold-water cures, a trip to the mountains, and, in anæmic in-

dividuals, to mild iron waters; in addition, quinine, arsenic, and belladonna. Local galvanization of the stomach, or the application of electricity to the central nervous system, may be attended with excellent results.

APPENDIX.

Leyden has called attention to the occurrence of periodical vomiting associated with pain, which occurs as a pure neurosis in anæmic and nervous individuals.

Under the term nervous gastroxynsis, Rossbach describes a symptom complex which occurs chiefly in individuals suffering from nervous exhaustion. It occurs every week or month in attacks of one to three days' duration. There is usually an excessive formation of acid in the stomach, nausea, and vomiting. The patients suffer from a feeling of dulness in the head and severe headache. Considerable lactic and hydrochloric acid can be demonstrated in the contents of the stomach. The attack subsides rapidly after the stomach is relieved of its contents, and is often aborted if lukewarm water is taken at the beginning. In addition to general treatment, we should order one to two glasses of lukewarm water or weak tea, since the disease seems to be the result of irritation of the sensory nerves of the stomach by an excessively acid gastric juice.

PART IV.

DISEASES OF THE INTESTINES.

1. *Acute Intestinal Catarrh. Enteritis Catarrhalis Acuta.*

I. ETIOLOGY.—Acute intestinal catarrh is an extremely frequent disease. It is sometimes independent and primary; sometimes it is secondary, and develops during the course of other diseases. Errors of diet constitute the most frequent cause of the primary form of the disease.

We see every day that excessive meals give rise to acute enteritis. The secretions of the digestive tract are evidently unable to properly digest the food, so that the latter undergoes abnormal decomposition, irritates the mucous membrane of the intestines, and gives rise to inflammation.

In other cases, it is the result of poor quality of the food. Enteritis is produced not infrequently by bad drinking water. Sometimes it is the result of the ingestion of spoiled food, such as spoiled meat, sour milk, unripe fruit, etc. In the summer months, a sort of epidemic may occur as the result of causes of this character.

A cold which may act either generally or locally not infrequently gives rise to intestinal catarrh.

Sudden wetting when the body is heated, or a cold bath, is often mentioned as the cause of acute enteritis. Among the local causes of this character, we may mention the drinking of cold water when the body is heated. In not a few cases, I have seen diarrhœa develop from the application of an ice-bag to the abdominal walls.

In rare cases, the disease is the result of injury, such as a fall, or blow upon the abdomen. Those cases in which a sort of internal injury has been sustained are much more frequent (ingested foreign

bodies, concretions in the intestines, worms, or foreign bodies which have been pushed into the rectum through the anus). Obstinate constipation gives rise not infrequently to enteritis, because the excessively hard masses of feces mechanically irritate the mucous membrane of the intestine.

Finally, we must mention toxic enteritis, which may be produced by various drastics, tartar emetic, arsenic, acids, alkalies, or caustics in general.

Acute enteritis occasionally develops epidemically, without our being able to demonstrate any of the previously mentioned causes. This occurs almost always in the hot summer months, especially if there has been a long heated term or marked changes of temperature. Such epidemics are observed occasionally as the forerunners of Asiatic cholera and dysentery.

In secondary acute enteritis, the inflammation is often propagated from adjacent parts. Thus, gastritis extends not infrequently to the mucous membrane of the small intestine. In other cases, the connection between acute gastritis and enteritis is somewhat different, inasmuch as decomposed or imperfectly prepared food is conveyed to the intestines, and there produces irritation. Acute enteritis also occurs with extreme frequency in peritonitis from the spread of the inflammation from the serous membrane to the mucous membrane of the intestine. Inflammation of the rectum occasionally occurs in women who suffer from gonorrhœa, because the infectious secretion enters the anus.

Sodomy may also give rise to inflammation of the rectum. In some cases, eczema around the anus extends to the rectum, and there produces catarrh of the mucous membrane.

In many cases, acute enteritis is the result of other diseases of the intestinal canal, such as ulcerative processes, invagination or volvulus, etc.

The disease is often the result of disturbances of circulation, especially of obstruction in the portal system. Tumors of the abdominal organs will produce a similar effect if the mesenteric veins are compressed. The obstruction to circulation is sometimes situated above the diaphragm (chronic diseases of the respiratory or circulatory organs).

General diseases occasionally give rise to acute enteritis. This is observed in cachectic conditions, for example, phthisis, Bright's disease, syphilis, etc. Or it occurs during infectious diseases. It is well known that acute enteritis plays a prominent part in typhoid fever, cholera, and dysentery. It is also observed in fibrinous pneumonia, pyæmia, septicæmia, etc. The septicæmic intestinal catarrhs include those which are observed in patients suffering from pulmonary gangrene and putrid bronchitis, as the result of swallowing the ichorous sputum. Some cases depend upon malaria. This is shown by their periodical and paroxysmal development and by their disappearance after the use of quinine.

In a number of cases, the symptoms of acute enteritis have been observed after extensive burns of the skin. The connection between the two affections is still obscure.

II. ANATOMICAL CHANGES.—When the anatomical changes are very marked, the mucous membrane of the intestine is very red and swollen. The redness is usually most marked upon the villi and the folds; sometimes it is uniform, sometimes in streaks or patches; extravasations of blood may be observed in some parts. The swelling of the mucous

membrane generally extends to the submucous tissue, where it is the result of an inflammatory, serous infiltration.

As a rule, the solitary and agminated lymph follicles are enlarged. They project to a more marked extent above the mucous membrane, and appear as gray, transparent granules or nodules. On being punctured, they discharge a little drop of almost clear fluid, and then usually collapse. When the acute enteritis has lasted for some time, no fluid may escape, and the follicle may not collapse after puncture. Hence the enlargement of the follicle depends at first upon inflammatory œdema, while later it is the result of inflammatory hyperplasia of the cellular elements.

These follicles are surrounded by an areola of enlarged blood-vessels.

The mesenteric lymphatic glands are almost always enlarged, streaked with blood, injected, and very succulent.

The muscular and serous coats are unchanged as a rule, except in secondary enteritis when affected by the primary disease. Sometimes unusual congestion is found in various portions of the serous layer.

In extremely rare cases, acute enteritis gives rise to losses of substances in the mucous membrane. These are sometimes superficial, so-called catarrhal erosions, sometimes more deeply spreading catarrhal ulcers. If the latter is the result of ulceration of the lymph follicle, it is known as a catarrhal follicular ulcer; if produced by destruction of the mucous membrane proper, as catarrhal ulcer of the mucous membrane. The latter begin generally as erosions.

Desquamation of the epithelial covering of the mucous membrane is found not infrequently in the dead body, but a part of these changes may be of a cadaverous character, probably from post-mortem maceration.

A part of the lesion disappears not infrequently in the dead body. This is particularly true of the congestion and swelling of the mucous membrane. Under such circumstances we must pay attention to the enlargement of the lymph follicles, the congested zone around them, the enlargement and injection of the mesenteric glands, and the character of the intestinal contents.

III. SYMPTOMS.—The symptoms are not always alike, and depend upon the part of the intestines in which the inflammation is situated. We therefore distinguish duodenitis, jejunitis, ileitis, typhlitis, colitis, and proctitis.

The inflammation almost always spreads from one part of the intestines to the adjacent part. Inflammation of the ileum and colon is most frequent, and this will first be considered.

The most constant symptom is diarrhoea. The patient must go to stool more often than normally, and discharges thin unformed masses. The number of evacuations may far exceed twenty in the twenty-four hours. In many cases the amount passed is greater than we would be led to expect from the quantity of food ingested.

Hence the evacuations are not only the result of unusually active peristalsis, but there is an excessive secretion of the mucous membrane in consequence of the inflammation. The appearance of the stools is very often abnormal. They may be bright yellow, greenish, tinged with blood, and sometimes contain an unusual amount of mucus. Their color sometimes changes after exposure to the air. If the evacuations follow one another very rapidly, they may not be stained by bile, and colorless, grayish fluid masses make their appearance in which float de-

squamated shreds of epithelium (rice-water stools). The fæcal odor then diminishes, and the stools have a peculiar sickish odor which has also been compared to that of semen. The patients sometimes pass very frothy and foul-smelling masses, in which decomposition and fermentation continue outside of the body. The consistence of the stools may be like that of thick or thin porridge, or they may be watery. The reaction is usually alkaline. The stools sometimes contain undigested particles of food, worms, or particles of fæces of extreme hardness; the latter indicate previous constipation. Under the microscope they are found to contain muscular fibres, starch granules, vegetable cells, and drops of fat. Bacteria form an important constituent, but pathogenic schizomycetes have not hitherto been discovered. In addition, we find desquamated epithelium cells, also round cells and a few red blood-globules.

We often find crystals of the triple phosphates, the neutral phosphate of lime, tablets of cholestearin, Charcot-Neumann crystals (vide Vol. I., page 233, fig. 58), and needles of the fatty acids.

Diarrhœa is sometimes the sole symptom of acute enteritis. It is often preceded by borborygmus, a feeling of distress, and cutting pain.

Physical examination of the abdomen may reveal extremely slight changes. The unusually vigorous peristaltic movements are sometimes recognized through the abdominal walls, loops of intestines rising and falling in places, to reappear in other parts. In other cases we find distention of the abdomen. This is the result of abnormally active development of gas in the intestines. The abdomen is often as tense as a drumhead, the diaphragm is pushed high up in the thorax, compressing the lungs and interfering with their mobility, and the apex of the heart is sometimes pushed into the fourth intercostal space. It is not astonishing, therefore, that many patients complain of palpitation, dyspnœa, and asthmatic disturbances. Flatus is often passed in abundance, after which the patient feels relieved. At the same time small quantities of a thin stool may be voided. On palpation we not infrequently notice a purring murmur, gargouillement, produced by pressure upon the intestinal fluid which is mixed with air. If pain is present, it is not infrequently ameliorated by increasing pressure upon the abdominal walls. The results of percussion vary, according as we percuss intestines which contain gas or are filled with non-aërated contents. (In the former event a tympanitic or metallo-tympanitic note, in the latter a dull percussion note is heard.) The rumbling intestinal noises may often be heard at a distance of several paces.

The excretion of urine is generally very scanty, and if the diarrhœa is very violent, almost complete anuria may result. The urine is concentrated, very acid, and on cooling, deposits urates; micturition is sometimes attended with burning pains in the urethra.

Fischl found casts in the urine not infrequently, and occasionally in very large numbers. They were usually hyaline, sometimes covered with epithelium and round cells, rarely with red blood-globules. The casts sometimes appeared very soon after the beginning of the diarrhœa, particularly in old persons, and if the evacuations were very copious. They soon disappeared from the urine. Albumin was sometimes present, sometimes absent. Fischl attributes the albuminuria to an aborted nephritis, the result, in great part, of diminished arterial pressure in the kidneys. This writer has also called attention to enlargement of the spleen.

Fever may be absent, but in some cases the temperature rises beyond

39° C. If the rise of temperature continues for several days, the diagnosis between simple acute enteritis and typhoid fever may be doubtful. In children and very excitable individuals, the fever may give rise to delirium and convulsions. At times it begins with a chill or chilly feeling.

Thirst is almost always increased. The appetite may be retained, particularly when the inflammation affects chiefly the large intestine. As a rule, however, the appetite is diminished or entirely abolished.

The signs of collapse are sometimes prominent. The skin becomes cool, and is covered with a cold, clammy sweat; the pulse is small and frequent; the eyes are sunken, and the voice becomes muffled. This is observed particularly in old individuals, or when the pains in the abdomen attain a certain severity, because some patients are extremely susceptible to them. Each attack of pain may be attended with syncope and symptoms of collapse.

The disease often lasts only one to two days, sometimes a week or more. The patients often feel very weak for a few days after recovery.

The symptoms described are observed particularly in ileo-colitis, the most frequent form of intestinal catarrh. When the acute catarrh is confined to the small intestines, diarrhoea is sometimes absent. The patients complain of rumbling and pain in the belly, but no changes are noticed in the evacuations.

Catarrhal inflammation of the duodenum is often attended with jaundice, because the opening of the ductus choledochus is occluded by secretion or by swelling of the mucous membrane, or the catarrh spreads directly from the mucous membrane of the duodenum to that of the ductus choledochus.

Jejunitis and ileitis cannot be differentiated during life; moreover, the jejunum and ileum are usually affected at the same time.

Isolated inflammation of the cæcum and vermiform appendix (typhlitis) occurs not infrequently. The most frequent causes are faecal stasis and foreign bodies. The chief symptoms are pain in the right iliac fossa, the demonstration of a tumor in this region, and dulness on percussion. Acute colitis is the most frequent form of intestinal catarrh, but is generally associated with ileitis.

If the rectum is inflamed (proctitis), violent tenesmus is usually produced. The patients feel the necessity of going frequently to stool, but very small quantities are evacuated. Shortly before the evacuation, cutting pain is felt in the left iliac region, and the evacuation itself is apt to be accompanied by the most violent pain. The left iliac fossa and pelvis are sensitive to pressure, and a dull or dull tympanitic note is heard on percussion. As a rule, the stools contain a very large amount of mucus, and often specks of blood, or even larger accumulations of blood.

The anus is often seen to undergo spasmodic contractions, and sometimes it is strongly retracted. Digital examination often produces an unusually severe pain. The finger is grasped spasmodically by the sphincter, and at the same time twitching movements can be felt. The mucous membrane of the rectum is often very warm, appears loosened, and unusually slippery. By means of the rectal speculum, we can detect redness, swelling, and excessive secretion of the mucous membrane. If proctitis has lasted for some time, the sphincter may undergo a sort of paralysis. Fluid constantly trickles from the anus, irritates the external integument, and produces an eczema intertrigo; prolapsus recti occurs not infrequently. The inflammation sometimes spreads to the

surrounding connective tissue, and gives rise to periproctitis and its sequelæ, especially fistula. Extensive enteritis may be associated with gastritis.

IV. DIAGNOSIS.—As a rule, the diagnosis of acute enteritis is not difficult. If it occurs during an epidemic of Asiatic cholera, and we must decide whether the diarrhœa is the result of a simple intestinal catarrh, or of mild cholera infection, the stool should be examined for Koch's comma bacillus (vide Vol. IV., sec. Cholera).

V. PROGNOSIS.—The prognosis is almost always good. As a rule, there is no danger unless the disease occurs in exhausted individuals. But the prognosis also depends upon the causation. If it is the result of disease of the liver, respiratory or circulatory organs, the individual attack may be relieved, but there is danger of relapse and the gradual development of chronic intestinal catarrh.

VI. TREATMENT.—If fever is present, the patients should remain in bed. If they complain of pain, a warm poultice may be applied to the abdomen. They should be permitted to take nothing but soup (strained oatmeal or barley soup).

Bouillon is also allowed. The patient may drink claret, either pure or diluted with water. Brandy and water may also be recommended. If the water is impure, it should be boiled for a little while in order to destroy the organisms and noxious substances contained in it. These measures sometimes prove sufficient.

Laxatives are indicated if the disease has been preceded by over-feeding or constipation, or the intestine contains foreign bodies or worms, or is filled with fermenting and decomposed masses. The simplest remedy is castor oil, of which one to two tablespoonfuls may be taken, mixed with the froth of beer to disguise the taste. A single large dose of calomel is also effective (℞ Hydrarg. chlorid. mite, pulv. jalap., sach. alb., āā gr. iij.).

If the development of acute enteritis is dependent on worms, the calomel may be mixed with an anti-helminthic.

After the administration of a laxative, the patient usually passes a few thin stools, and then the disease often recovers spontaneously.

If the diarrhœa continues, we may order the following:

℞ Pulv. ipecacuanh. opiat.,
Sacch. alb. āā gr. ivss.
One powder every three hours.

℞ Tinct. opii,
Tinct. valerian. æther. āā 3 i.
M. D. S. 15 drops t. i. d.

℞ Bismuth subnitrat. gr. viij.
Opii. gr. ss.
Sacch. alb. gr. v.
One powder every three hours.

We may also mention the following astringents: Acetate of lead (gr. $\frac{3}{4}$ every two hours); tannic acid (gr. iss. every two hours); nitrate of silver (gr. iss. : $\frac{3}{4}$ iss.; glycerin, 3 iij.; one tablespoonful every three hours); alumen (gr. iss. every two hours); alumina acetica (gr. iss. : $\frac{3}{4}$ iij. every two hours), etc.

In catarrhal inflammation of the cæcum, colon, and rectum, local

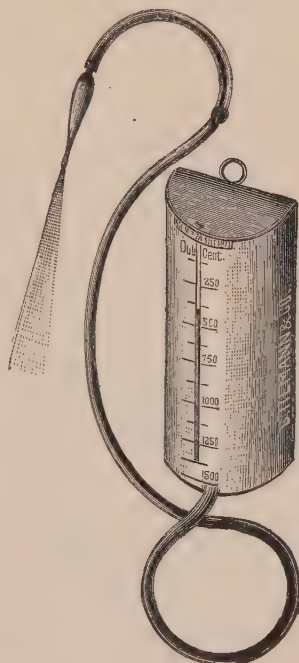
applications may be made. For this purpose, we employ a rectal tube, the posterior extremity of which is fitted with a rubber tube, about one to two metres in length, while the free end is provided with a glass funnel (vide Fig. 16). By means of this apparatus, large amounts of fluid can be poured into the large intestine when the patient is lying on his back; indeed, the fluid may pass the ileo-cæcal valve and enter the small intestine. The pressure of the fluid can be varied at will by raising or lowering the funnel. It should be remembered that in the beginning, as a rule, no water flows from the funnel into the rectum; the flow begins only when rapid pressure has been made a number of times upon the rubber tube, in order to remove air from the rectum.

Fig. 16.



Funnel apparatus for intestinal infusion.
After Hegar.

Fig. 17.



Apparatus for intestinal infusion. After Korup.

Korup recently constructed an apparatus which is shown in Fig. 17. It consists of an irrigator, the tube of which can be closed by a stopcock. The tube enters a rectal bougie. A glass tube, which is connected with the inside of the irrigator, shows the amount of fluid in the latter.

It is, perhaps, best to simply wash the rectum and then to follow with a weak solution of nitrate of silver (gr. iss. to viiss. : $1\frac{1}{4}$ pint of distilled water).

Certain prominent symptoms require special treatment. If the pains are very severe, we may make a subcutaneous injection of morphine. This often relieves the diarrhœa, especially if peristalsis has been very vigorous. Other narcotics have also been recommended to relieve

unusually active peristalsis, viz., chloral hydrate, potassium bromide, and strychnine. In violent tenesmus and spasm of the anus, we may employ suppositories of morphine or belladonna. (℞ Morphin. hydrochloric., gr. ss.; Ol. Cacao, q. s. ut fiant suppositoria No. iij. D. S. One suppository two or three times a day. ℞ Ext. Belladonnæ, gr. $\frac{1}{4}$; Ol. Cacao, q. s. ut f. suppositoria No. iij.)

The etiology must also be taken into consideration. When the diarrhœa is produced by malaria, it is rapidly cured by quinine (gr. xv.—xxx. every day in a single dose). Suitable treatment must also be adopted when the disease is associated with affections of the heart, the lungs, liver, etc.

As a rule the patients must be carefully watched during convalescence. Strict diet should be maintained for a considerable time, and sometimes a woollen abdominal bandage should be worn to prevent catching cold.

APPENDIX.

a. Cholera morbus (Cholera nostras, Gastro-enteritis catarrhalis acutissima).

1. This term is applied to an acute gastro-intestinal catarrh of extreme intensity, whose symptoms are very like those of Asiatic cholera. As a rule, the disease begins in the latter part of summer (July, still more frequently in August and September), when the oppressive heat during the day is followed by cooler temperature at night. Isolated or sporadic cases are sometimes observed, but the disease also occurs in a sort of epidemic.

Its causes are cold, errors of diet, ingestion of impure drinking water, spoiled meat or unripe vegetables. It is very probable that a certain part is played by infection with low organisms, but this has not been determined with certainty.

2. The first symptoms sometimes develop with extreme suddenness; sometimes there are prodromata, such as anorexia, nausea, and general malaise.

The first symptoms often occur at night. The patients awake, complain of a feeling of pressure in the epigastrium, feel anxious, then experience nausea and repeated vomiting. At first this expels the contents of the stomach, later biliary, yellowish-greenish, finally almost watery masses are vomited. At first the vomiting is usually easy. It occasionally occurs with such frequency that the patients vomit twenty to forty times within a few hours.

At the same time or soon afterwards, the patients feel a rumbling and pain in the belly, and diarrhœa begins. At first the stools contain masses of the consistence of thick porridge, but the fecal character is soon lost. The stools become colorless, contain shreds of desquamated epithelium, lose the fecal odor—in short, they acquire the characteristics of the dreaded rice-water stools, such as are observed in the course of Asiatic cholera. The diarrhœa sometimes is so frequent that the patients use the vessel almost constantly. It is often extremely copious, so that we must assume an unusually vigorous transudation of fluid from the blood-vessels and a hyper-secretion of the intestinal mucous membrane.

The clinical history acquires an almost specific character, from the symptoms of severe collapse which soon make their appearance. The skin is cool, sometimes covered with cold, clammy sweat, appears flaccid,

and occasionally can be lifted into permanent folds. The features are sunken, so that after a short time they are hardly recognizable. The nose becomes pointed, the eyes are situated deep in their sockets, and usually surrounded by a grayish ring, the expression is dull and apathetic, the eyelids are often half closed and the globe rolled upwards. The pulse is strong and extremely frequent, the heart sounds are very feeble.

The patients speak in a feeble muffled tone, and the voice may sometimes become falsetto.

They complain of unquenchable thirst, throw themselves restlessly about, and often cry out suddenly on account of painful muscular spasms. These are observed most frequently in the calves, more rarely in the thighs, arms or abdominal muscles. Involuntary contractions of the muscles occur during the cramps.

Diuresis is very much diminished or almost abolished. The urine often contains hyaline casts and albumin, and an extremely large amount of indican.

As a rule, the disease does not last more than one to two days. The vomiting and diarrhœa diminish and finally cease, the skin becomes warm, the pulse stronger, the muscular pains disappear, the voice resumes its former timbre, a profuse excretion of urine occurs, and recovery ensues. Great feebleness often remains for days and weeks. A fatal termination hardly ever occurs except in children, old people, or other exhausted individuals.

If the disease takes a fatal turn, the vomiting and diarrhœa usually cease, but the patients complain of distressing singultus, collapse increases, the pulse and heart sounds become imperceptible, and finally death occurs.

3. The diagnosis is usually easy; since Koch showed that the comma bacillus is always found in the stools in true Asiatic cholera, we can readily make the differential diagnosis between this and cholera morbus by the aid of the microscope. (For further particulars vide Vol. IV., Asiatic Cholera.)

The symptoms of cholera morbus may sometimes be produced by poisoning by tartar emetic, arsenic, and corrosive sublimate, so that in suspicious cases the gastro-intestinal contents may be subjected to a chemical examination.

4. The preparations of opium enjoy great repute in the treatment of this disease: \mathcal{R} Opii, gr. ss.; sacch. alb., gr. v. M. f. p., d. t. d. No. x., one powder every two or three hours. \mathcal{R} Tinct. Opii, Tinct. Valerian., \mathcal{A} ther, ââ $\frac{3}{4}$ ss.; fifteen drops t. i. d. \mathcal{R} Pulv. Ipecacuanh. co., Sacch. alb., ââ gr. v.; one powder every two to three hours.

In addition, the abdomen should be covered with a warm poultice, and hot bottles applied to the side of the body, if the skin feels cool and the patient complains of cold. The bottle should be covered with cloth, and not be so hot as to produce burns. Violent vomiting may be relieved by a subcutaneous injection of morphine in the epigastrium. In addition, pieces of ice may be given to diminish the thirst and vomiting. The muscular spasms can also be relieved most effectually by injections of morphine. In addition, the parts may be rubbed with cloths or spirits of camphor. To combat the collapse we may order brandy, port wine, champagne, in teaspoonful doses, or inject camphor subcutaneously (Camphor, gr. xv.; \mathcal{O} l. amygdal., 3 iij.; one syringe-ful t. i. d.).

Bouillon is the only food which may be allowed, and even after recovery the patient must exercise great care in diet for some time.

b. Acute gastro-intestinal catarrh of infants.

1. ETIOLOGY.—The great mortality in children, particularly in large cities and during the summer months, is almost entirely owing to this disease.

It seems to be the result of errors in nutrition, of certain developmental processes, or of certain miasmatic influences.

Milk, the most natural article of food for infants, is very apt to undergo changes and to give rise to gastro-enteritis. In addition, the intestinal tract of nursing infants is extremely sensitive. This hypersensibility is recognized by the fact that acute gastro-enteritis may be produced in infants at the breast as the result of mental excitement in the mother, although no change in the milk can be discovered in such cases.

It should not be forgotten that the organization of the digestive apparatus in infants constitutes a predisposition to disease. Very little saliva is secreted, the fundus ventriculi is still imperfectly developed, and the muscular coat of the stomach does not possess much power. The principal cells of the stomach glands are but little developed, so that the gastric juice is poor in pepsin. Furthermore, the pancreas furnishes no glycoënic ferment until the end of the third month of life.

Experience teaches that certain processes of development in infancy favor the development of acute gastro-enteritis. This category includes teething and weaning. In very many children the symptoms of acute gastro-enteritis occur with the eruption of each tooth. Diarrhœa is most apt to occur at the period of weaning, if the transition to mixed diet is undertaken too suddenly. Another very important form of acute gastro-enteritis is that which occurs in the hot summer months in the form of an epidemic, and is known as cholera infantum or summer diarrhœa. It is most frequent in August and September, more rare in July and October. If we bear in mind that the disease is found principally in badly ventilated and overcrowded houses, we will be inclined to assume the existence of miasmatic influences in this disease.

II. ANATOMICAL CHANGES.—The anatomical changes differ in no respect from those observed in acute gastro-enteritis of adults. In cholera infantum we often notice softening of the stomach (vide page 96). If the children have perished with choleriform symptoms, the blood often has a dark color and increased consistence, and the serous membranes are peculiarly dry, or present a soapy appearance.

III. SYMPTOMS.—The chief symptoms consist of vomiting and diarrhœa. Sometimes one, sometimes the other begins first. At first the milk is vomited in caseous lumps. Later, it is thin and uncoagulated, because the stomach no longer produces sufficient acid to coagulate it. As collapse increases, the vomiting subsides, and singultus takes its place.

The diarrhœa is sometimes so frequent that more than forty evacuations occur within the twenty-four hours. The yellow color of the normal stool is mixed with greenish parts, or the stools are entirely greenish, or they are yellow at first and become green after exposure to the air. Later they are apt to lose the biliary coloring, so that we find chiefly a watery fluid, at the most a few grayish, yellowish, or greenish specks and masses. Sometimes the stools become more consistent for a while, and occasionally have a brownish color, but often a very foetid smell. So long as the stools are not watery, they usually contain whitish or grayish-white shreds, which have been regarded as undigested frag-

ments of casein, but Uffelmann showed that these are composed of drops of fat, or of bacteria, or of crystals of a combination of fatty acids and lime. The reaction of the *fæces* is almost always acid. The evacuations are often preceded by colicky pain; the children cry out aloud, and draw their legs upon the abdomen. The pains are not infrequently relieved by pressure on the belly.

The abdomen is sometimes sunken, sometimes distended by gas in the intestines. Palpation sometimes reveals a peculiar sensation of fluctuation which corresponds to distention of the intestine with fluid. If the anus and thighs are soiled by the *fæces*, eczema intertrigo is often produced. Prolapsus of the rectum is occasionally observed.

The appetite is often undisturbed, but the patients soon vomit the ingested food. Thirst is increased to an extreme degree.

The excretion of urine often ceases. The mucous membrane of the mouth may be dry and hot, and covered with masses of sprue or with aphthæ.

If the disease, which occasionally ceases in one or two days, lasts for several days, the symptoms of severe collapse make their appearance. The extremities become extremely cold, while the trunk feels very hot. The fontanelles are depressed, the eyes are sunken in, and deep wrinkles appear in the face, which looks like that of an old man.

The child becomes apathetic, and often lies with half-closed eyes; the eyelids are partly adherent by dry mucus, the cornea dries, may ulcerate, and even undergo perforation.

Edema sometimes develops, and extravasations of blood may occur into the skin and mucous membranes. Sometimes we notice the signs of objective dyspnœa, which have been attributed usually to thickening of the blood, but which Gerhardt explains by anæmia of the medulla oblongata. Death sometimes occurs after cerebral symptoms, convulsions, muscular twitchings, delirium. This symptom-complex has also been called hydrocephaloid, because it is similar to the symptoms of acute hydrocephalus. Among the complications may be mentioned atelectasis of the lungs, broncho-pneumonia, and thrombosis of the sinuses. In favorable cases the rapidity of recovery is often astonishing. Children who to-day are struggling with death, to-morrow appear almost well.

IV. DIAGNOSIS.—The diagnosis of the disease is easy, but is only completed when the etiology is recognized. The physician should investigate in detail the mode of nourishment, and should himself examine the various articles of food.

V. PROGNOSIS.—The prognosis is always serious, but of all the forms of gastro-enteritis, summer diarrhœa is the most dangerous.

VI. TREATMENT.—We will first give a brief resumé of the principles of rational nutrition of infancy. Nursing at the breast is the most natural and the best form of nutrition, but mothers who have not reached the age of eighteen years should not be allowed to nurse their children at the breast. We know from experience that their milk is not sufficiently nutritious and that lactation in very young mothers threatens the production of chlorosis and phthisis. Nor should nursing at the breast be carried on by mothers who present an hereditary family history of phthisis, cancer, or neuroses of all kinds. This is also true of mothers who suffer from chlorosis or infectious diseases. A syphilitic mother should not be allowed to nurse her healthy infant because the latter may be infected in this manner (vide Vol. IV., Congenital Syphilis). Nursing

is sometimes prevented by purely mechanical factors, for example, a poorly developed nipple which cannot be grasped by the child. As a matter of course, nursing must cease if the milk is produced in insufficient quantities.

During the first few months of life, the child should be nursed every two hours, later every three hours, the breast being first offered about six o'clock A.M., and not later than ten o'clock P.M. After each nursing the mouth should be carefully washed with a piece of linen which has been dipped in cold water, in order to prevent the retention and decomposition of the particles of milk in the mouth. During the period of nursing the mother should avoid excitement and also coitus, because these are apt to be followed by symptoms of gastro-enteritis in the infant. The mother should avoid fresh vegetables and acids as much as possible. Beer, eggs, milk, and farinaceous articles may be especially recommended. When pregnancy occurs, nursing should be discontinued because the mother's milk becomes poorer, and in addition the maternal organism is incapable of properly fulfilling both functions. It is sometimes found that the children do not thrive, although nothing abnormal can be detected in the mother. Some other mode of nourishment must be then adopted. However, the growth of the child cannot always be determined with the eye, and we must resort to the use of the scales. The child should be weighed once a week under exactly similar conditions. Immediately after birth the average weight of the healthy new born infant is 3,250 gm.

Boys weigh about 120 gm. more than girls, and the children of a multipara 60 to 120 gm. more than those of a primipara. During the first three days after birth the weight diminishes about 300 gm. on account of the losses in the meconium, desquamation of the skin, and respiration. The weight then slowly increases, and on the tenth day reaches the same figures as immediately after birth. During the first five months the infant increases 20 to 30 grammes daily, later from 10 to 15 grammes. The following table indicates the weight of the infant during the first year:

Weight at birth, 3,250 grammes.

Age.	Daily increase in weight in gm.	Monthly increase.	Weight.
1st month.....	25	750	4,000
2d "	23	700	4,700
3d "	22	650	5,350
4th "	20	600	5,950
5th "	18	550	6,500
6th "	17	500	7,000
7th "	15	450	7,450
8th "	13	400	7,850
9th "	12	350	8,200
10th "	10	300	8,500
11th "	8	250	8,750
12th "	6	200	8,950

If for any reason the mother is unable to nurse the child, a wet-nurse furnishes the best substitute. In selecting a wet-nurse, we should be guided by the same principles as in determining whether the mother should be permitted to nurse her child. The difference in age between the mother's child and that of the wet-nurse should not exceed two months.

When nursing at the breast is impracticable, we must resort to nutrition with animal milk or substitutes for milk. The chemical constitution of mother's milk is compared with that of animals in the following table:

	Mother's milk.	Asses' milk.	Cow's milk.	Goat's milk.
Water.....	889.08	890.12	864.06	844.90
Solid matters	110.92	109.88	135.94	155.10
Casein.....	39.24	35.65	55.15	55.14
Fat.	26.66	18.53	86.12	56.87
Milk sugar.....	43.64	50.46	38.03	36.91
Salts.....	1.88	5.24	6.64	6.18

Asses' milk resembles mother's milk more closely than other varieties, but practically we may only choose between cow's milk and goat's milk. The former is preferable, but water and sugar should be added to it, in order to make it resemble mother's milk more closely. It is important that those who superintend the feeding of the infant, should know that red litmus paper is quickly turned blue by mother's milk, and that lime-water should be added to cow's milk, until the reaction produced by it upon litmus paper approximates that produced by mother's milk. Cow's milk sometimes has an acid reaction, even when taken directly from the udder.

The milk should be boiled as soon as received, and then one to two tablespoonfuls of lime-water added, particularly in summer, in order to avoid acidification. It should always be given to the infant at the temperature of the body. It may be warmed by placing a bottle of milk in a vessel of hot water. When it has reached the proper temperature, a sufficient amount of lime-water should be added, and then enough water to dilute the milk by half (during the first month). The dilution should be gradually diminished so that the child takes pure milk after the fifth month. In addition, the milk should be well sweetened with sugar.

The milk should be given in the same way, with regard to quantity and time of administration, as has been recommended with regard to nursing at the breast. After being used, the bottle and nipple should be carefully washed in salt and water, and then kept in pure water until again required. The children should not be permitted to keep the nipple in the mouth as a "soothing measure."

But despite all these precautions, the digestion of cow's milk is often difficult, as is shown by the fact that the feces of children who are fed with cow's milk have a cheesy, whitish-yellow appearance, while in children who are nursed at the breast, the feces have a uniform yellow color. This difference is still more marked when the infant is fed with goat's milk.

Artificial feeding with substitutes for milk is only indicated when good milk cannot be obtained, since it favors the development of diarrhoea and rachitis. These substitutes may also be employed temporarily in gastro-enteritis. We may recommend Nestlé's food, Biedert's cream mixture, Swiss condensed milk, Liebig's infant's food, Löfflund's food, and Beneke's leguminous soup.

Healthy children should be gradually weaned at the age of nine months. Weaning should be delayed if the child is teething, or if infantile diarrhoea is rife in the neighborhood, or during the hot summer months. The milk should be gradually mixed with weak bouillon, later bouillon mixed with egg may be given at mid-day, and a very soft boiled egg may be given in the morning or afternoon; also cocoa, crackers, or wine. Finally, we may give raw scraped meat or ham.

If gastro-enteritis has developed, no milk should be given to the child for twelve to twenty-four hours. In its stead we may order water which has first been boiled, and then allowed to cool, mixed with claret or brandy. The milk should be permanently withheld, if it was not well tolerated previously or if the symptoms do not diminish in three to four days despite proper treatment. In its stead, we may give Nestlé's or Faust's food, barley-water, sago, or arrow-root. Raw scraped meat or mutton, and beef-tea are often very useful. Beef-tea is prepared in the

following manner: Fresh lamb or beef is cut into small pieces, eight times the quantity of water is added and allowed to stand for a half an hour. It is then covered, placed in a bottle in hot water and the latter allowed to boil. The meat is then squeezed, the fluid strained through a cloth, and a little salt or sugar added. It should be prepared fresh three times a day. Among medicinal agents, antifermentatives produce the best effect. We prefer calomel (gr. $\frac{1}{4}$ every two hours); in violent vomiting creasote (gr. $\frac{3}{4}$, spirit dilut. gtt. x., decoct. rad. salep. $\bar{\text{z}}$ iiiss., one teaspoonful every two hours). Carbolic acid (gr. iss. to gr. viiss.: $\bar{\text{z}}$ ij., one teaspoonful every two hours), resorcin (gr. viiss.: $\bar{\text{z}}$ iiiss., one teaspoonful every two hours), and benzolum have also been recommended. If the diarrhœa is very violent, the administration of astringents must be resorted to. The best in our opinion is nitrate of silver (gr. viiss., aq. destil. $\bar{\text{z}}$ iij., glycerin $\bar{\text{z}}$ ss., one teaspoonful every two hours). With regard to other remedies we refer to page 110. Preparations of opium should not be given to nursing infants. This is often done perhaps without bad effects, until finally poisoning is produced despite every precaution, as we have observed in a number of cases.

We may also recommend treatment with enemata of nitrate of silver (gr. iij. to gr. viiss. at a dose). Hydropathic compresses to the abdomen are often employed, if the temperature of the trunk is very much increased. Small doses of hydrochloric acid (gtt. viij.: $\bar{\text{z}}$ iijss., one teaspoonful every two hours) are occasionally indicated when the vomiting of uncoagulated milk indicates a deficiency of acid in the stomach.

Threatening collapse may be combated by subcutaneous injections of camphor (gr. xv.; Ol. amygdal. $\bar{\text{z}}$ iij. M. D. S. One-quarter of a syringe-ful); champagne and port wine.

2. Chronic Intestinal Catarrh. *Enteritis Catarrhalis Chronica.*

I. ETIOLOGY.—Chronic enteritis either develops as such from the start, or it follows acute enteritis if attacks of the latter recur with unusual frequency, or appear before the previous attack has entirely subsided. In the latter cases the etiology of chronic enteritis is the same as that of the acute form. But even that form which runs a chronic course from the start is hardly ever the result of other causes, unless certain etiological factors have a tendency to favor the development of chronic rather than of an acute intestinal catarrh. These include particularly conditions of stasis (diseases of the portal vein, hepatic diseases, chronic affections of the respiratory or circulatory organs) and general diseases (pulmonary phthisis, malaria, and cachectic diseases). It must also be mentioned that the presence of infusoria in large numbers in the large intestines keeps up, if it does not give rise to, a chronic catarrh.

II. ANATOMICAL CHANGES.—As a rule, the color of the mucous membrane is livid or reddish-brown, and the large veins are generally unusually full. If the catarrh has lasted for a long time, the mucous membrane not infrequently has a slate-gray color, owing to the fact that the blood pigment in previous extravasations of blood is converted into melanin. This is generally most abundant at the apices of the villi and around the lymph follicles.

The mucous membrane and submucous tissue are often thickened, and many of the lymph follicles are enlarged. In some cases the increased thickness is the result of hyperplasia of the connective tissue.

The muscular coat may also take part in the hyperplastic process. It is thickened three to four fold, and traversed by fan-like bands of connective tissue. The serous layer in places presents tendon-like thickenings and opacities. The tissue of the mucous membrane sometimes undergoes hyperplasia to such an extent as to give rise to the formation of polypi (enteritis polyposa). Lebert describes a case in which numerous polypi were present throughout the entire large intestine. Occlusion and cystic dilatation of the glands are sometimes observed, and are associated occasionally with the formation of polypi (enteritis polyposa cystica).

Hyperplasia of the muscular coat sometimes produces stenosis. This is observed most frequently near the ileo-cæcal valve, but also at the sigmoid flexure and at the outlet of the rectum. In the chronic enteritis of children, however, the mucous membrane is not infrequently pale, and the intestinal walls are remarkably thin and atrophic. Atrophic changes in the glandular apparatus may also be observed under the microscope.

The hypersecretion of the mucous membrane gives rise to the formation of a serous, sometimes of a mucoid or pus-like fluid, which is found upon the inner surface of the membrane. Among the sequæ of chronic enteritis the most important are ulcerative processes of the mucous membrane. One form is the result of direct ulceration, the other is produced by suppuration of the lymph follicles. The former may be called catarrhal ulcers of the intestine, the latter follicular ulcers.

In catarrhal ulceration the changes generally begin with superficial epithelial losses of substance. These are originally round or lentil-shaped and gradually spread in depth and circumference. On account of the coalescence of adjacent ulcerations the ulcer finally becomes irregularly jagged. The adjacent mucous membrane is often undermined. Strips of mucous membrane sometimes extend into the ulcer from its edges, and occasionally isolated islets of mucous membrane remain within the ulcer. Polypoid proliferations are sometimes noticeable at the edges.

There are numerous terminations and complications of catarrhal intestinal ulcers. The least important is the formation of pus which attends the development of the ulcer. More serious dangers arise when larger vessels are involved in the destructive process, since violent hemorrhage may then be produced. In other cases the destructive process spreads deeper and deeper and gives rise to perforation and perforation-peritonitis; or if this occurs in parts of the intestines which are destitute of a peritoneal covering, fæcal abscesses develop. Under certain circumstances these may extend to the cervical region, and death occurs after symptoms of pyæmia. Perforation is prevented not infrequently by the formation of adhesions to adjacent loops of intestine or to other organs; or an encapsulated peritonitis is produced, into which the perforation first occurs. If perforation of intestine finally occurs, despite previous adhesions, abnormal communications are formed, for example, with other loops of intestine, the bladder, vagina, stomach, or gall-bladder, or large abdominal vessels may be eroded (aorta, vena cava, portal vein, etc.). If the adhesion has formed between the intestine and the anterior abdominal wall, an external intestinal fistula may be produced. Ulcers in the rectum may give rise to inflammation of the surrounding cellular tissue (periproctitis).

The most favorable termination is cicatrization. The cicatricial tissue is often pigmented and retracted to such an extent that the edges of the ulcer are almost in contact with one another. As a matter of course,

the lumen of the intestine is thus narrowed, and serious symptoms of intestinal stenosis may be produced.

Follicular intestinal ulcers are situated chiefly in the colon. They are sometimes so numerous that the mucous membrane appears to be perforated almost like a sieve; they may also be very numerous in the rectum. We have previously stated that the lymph-follicles become enlarged in enteritis. This enlargement is the result, at first, of an inflammatory serous infiltration, but later of a cellular proliferation. The mutual pressure of the cells first gives rise, in the middle of the follicles, to necrosis and cheesy degeneration, then to softening and rupture externally. The middle of the follicle at first appears opaque, yellowish and cheesy. Later we find a sharply-defined, crater-like ulcer with raised edges. The ulcerative degeneration continues to spread, soon passes beyond the solitary follicles and extends to the mucous membrane. The coalescence of adjacent ulcers gives rise to irregular but sharply-defined losses of substance, whose edges are undermined over considerable areas. The edge of the mucous membrane is often bent into the base of the ulcer. Vitreous, gelatinous masses are found not infrequently in the ulcers, similar to those which are observed in the stools during life. The complications are the same as those of simple ulcers of the mucous membrane.

III. SYMPTOMS.—Changes in the stool constitute the most important symptoms of chronic intestinal catarrh. As a rule, we notice constipation, or there is irregularity in the evacuations, so that constipation alternates with diarrhoea. Chronic diarrhoea is rarely observed, although cases are known in which it has lasted twenty years. Sometimes several thin stools are passed only in the morning, and many patients experience the desire to go to stool while still in bed. The evacuation sometimes contains undigested articles of food, not infrequently a large amount of pus and mucus. Indeed, at times, faecal matter is entirely absent in the passages, and these consist solely of mucus and pus. This is especially true of catarrh of the large intestine, particularly the rectum. A slight admixture of blood is sometimes observed.

The mucus sometimes forms cylindrical moulds of the intestine. These may be more than one-half metre in length, or they have a flattened, tape-like shape (enteritis membranacea). This sometimes occurs only at times, perhaps at intervals of months or years. Occasionally it continues for a long time; it is most frequent in middle life, rare in old people or children. In these moulds Da Costa found mucin and sometimes traces of albumin. Alcohol and carbolic acid produce retraction of the masses. Bjoernstroem found that they were composed of a structureless substance, which contained a few free nuclei, epithelium, and small drops of fat. This form of disease is found especially in hysterical and hypochondriacal individuals. The passage of the membranes is often preceded by violent pains and loud rumbling in the abdomen. Chronic intestinal catarrh of childhood is sometimes associated with stools which contain fat (diarrhoea adiposa.) The stools have a fatty gloss, smell strongly of fatty acids, and are grayish-yellow or reddish-gray in color. Under the microscope they are found to contain numerous large drops of fat. In healthy faeces the fat drops are scanty and small in size. Biedert found from 3.8–20.3 per cent of fat in the faeces of healthy infants, and 41.17–67 per cent in fatty diarrhoea. The latter seems to develop when the duodenum is affected to a marked degree and interferes with the passage of bile and pancreatic juice into the intestines, since these fluids cause

an absorption of fats in the intestine. Demme also found parenchymatous and interstitial inflammation of the pancreas. Fatty diarrhœa of children, however, should not be regarded as a special disease.

The microscopical examination of the *feces* should not be neglected in chronic intestinal catarrhs. A good idea of the impairment of digestion is easily obtained from the greater or lesser abundance of particles of food in the evacuation. The patients complain not infrequently of borborygmus and cutting pains in the abdomen, which appear sometimes after meals or shortly before an evacuation. Many patients also suffer from flatulence, which may be so severe as to produce disturbance of breathing, palpitation of the heart, and a rush of blood to the head. These symptoms are partly reflex, partly the result of mechanical interference with the movements of the diaphragm. The discharge of flatus usually produces great relief. In many cases my attention has been attracted by slowness of the pulse. If the inflammation extends to the stomach we also find coated tongue, a bad taste in the mouth, anorexia, singultus, etc.

Emaciation and pallor of the skin soon make their appearance. In addition, the patients are greatly worried about their condition, become dissatisfied with themselves and those about them, and fall into a condition of hypochondria and melancholy. The latter may become intensified into insanity.

Chronic enteritis may affect the entire intestine or single parts. In the latter event the colon and ileum are most frequently affected at the same time.

Chronic duodenitis is generally associated with chronic gastritis, and is recognized only by the accompanying jaundice.

Isolated ileitis cannot be recognized during life. The symptomatology given above holds good for the combination of ileitis and colitis.

Inflammation of the rectum (proctitis) not infrequently gives rise to tenesmus, though not so severe as in acute enteritis. The stools may be also entirely purulent (proctitis blenorrhoica). It may be associated with inflammation of the surrounding connective tissue, and sometimes gives rise to prolapse and eczema around the anus. Dilatation of the hæmorrhoidal veins is often observed.

Proctitis chronica and hæmorrhoids may be the results of the same primary disease, for example, cirrhosis of the liver; or the catarrh causes looseness of the mucous membrane, and thus favors the development of hæmorrhoids; or, finally, the dilatation of the rectal veins gives rise to proctitis.

Chronic enteritis lasts months and years; sometimes a lifetime.

The disease is dangerous to life in children and old people, rarely in vigorous adults. Children grow weak, emaciate, and finally die from dropsy and marasmus, sometimes from intercurrent bronchitis, bronchopneumonia or thrombosis of cerebral sinuses. In old people, death occurs with signs of increasing exhaustion. (Edema of cachectic character, or unilateral œdema from marantic thrombosis, sometimes appears as the precursor of death.

Catarrhal ulcers of the intestines are often latent during life. Nothnagel attaches great importance to the presence of pus in the stools. Great significance has also been attached to the appearance of swollen sago-like grains in the stools. These were regarded as an accumulation of intestinal secretion in the follicular ulcers. But, apart from the fact that these little lumps are also observed in simple enteritis, Virchow

showed that they are composed of swollen starchy matters from the food. The presence of shreds of the mucous membrane in the stools is a positive indication of intestinal ulceration, but this is a very rare event. Attention should also be paid to localized pain, circumscribed peritonitis, hemorrhages, and symptoms of perforation of the intestine. In rectal ulceration, mucus, pus, and blood will often adhere to the examining finger, and the fæces are not infrequently surrounded by these products. It should be remembered that diarrhœa is by no means present in all cases of ulceration of the intestines.

IV. DIAGNOSIS.—This is not difficult, except with regard to accurate localization. The diagnosis is not complete until the etiology has been recognized.

V. PROGNOSIS.—Chronic enteritis is often a very obstinate affection. Profound mental depression is an especially grave complication. The disease produces direct danger to life in children and old people. The prognosis is also unfavorable when the primary disease cannot be relieved.

VI. TREATMENT.—If constipation is present, we should order mild laxatives:

R Aloes,
Ext. rhei comp. āā gr. xxiiss.
Pulv. et succ. liq. q. s. ut fiant pil. No. xxx.
D. S. Two to four pills to be taken at night.

R Aloes,
Ext. rhei comp.,
Jalapæ. āā gr. xv.
Pulv. et succ. liq. q. s. ut fiant pil. No. xxx.
D. S. Two to four pills at night.

R Pulv. liq. comp. ʒi.
D. S. One to two teaspoonfuls at night.

Numerous other laxatives have been recommended, which it is unnecessary to mention. Many patients resort to household remedies: a glass of cold water every morning before breakfast, smoking a cigar early in the morning, stewed fruit, etc.

Many resort to the use of bitter waters, for example, Ofener (Hunyadi-Janos, Franz Josef, or Victoria), Püllnaer, Suidschuetzer, Friedrichshall, and Sedlitzer bitter waters. One to two wineglassfuls may be taken early in the morning; and half an hour later, a glass of cold water. In marked atony of the muscular coat of the intestines, I have seen good results, in a number of cases, from massage of the abdomen. Not infrequently obstinate constipation is relieved by faradization of the intestines (vigorous current, one electrode upon the lumbar spine, the other firmly pressed upon the abdomen, and applied labile for three or ten minutes; or one electrode is introduced five to ten cm. into the rectum, the other as before upon the abdomen; daily sittings). If diarrhœa is present, we may order the preparations mentioned on page 110.

A strict milk diet has produced recovery in a number of cases of diarrhœa which had lasted for years.

Mineral waters may be very useful in chronic intestinal catarrh. When obstinate constipation is present, we may recommend the alkaline saline waters (Marienbad, Franzensbad, Rohitsch, Steiermark, Tarasp). The salt waters of Kissingen and Homburg also merit favorable men-

tion. If there is a tendency to chronic diarrhœa, we may resort to the alkaline saline waters of Carlsbad, Ems, or Vichy, or the warm sodium chloride wells of Wiesbaden and Baden-Baden. In such cases, good results are sometimes obtained from the earthy mineral waters (Wildungen, Waldeck, Lippspringe).

Many patients are improved or cured by cold-water treatment, a trip to the mountains, or a sea voyage. The grape cure is not without benefit in many cases.

Due attention should be paid in treatment to the etiology. If infusoria are found in the stools we should make daily intestinal infusions with lukewarm water, and then inject a dilute solution of bichloride of mercury (gr. iss. : $\frac{3}{4}$ x.).

APPENDIX.

Phlegmonous inflammation of the intestine (enteritis phlegmonosa, s. submucosa, s. purulenta) possesses merely an anatomical interest. It gives rise to diffuse purulent infiltration, or to the formation of abscesses in the submucosa, sometimes in the muscular coat. In Bellfrage's case, in which the jejunum was affected, the diseased part was 18 cm. in length. Rupture externally results in peritonitis. If the process is situated in the rectum, it may be followed by periproctitis. The disease is usually secondary to typhoid, tubercular, dysenteric, or cancerous ulcers.

3. *Inflammation of the Cæcum and Vermiform Appendix and Surrounding Parts. Typhlitis, Perityphlitis, and Paratyphlitis.*

I. ETIOLOGY.—Inflammation of the cæcum and vermiform appendix is known as typhlitis, inflammation of the peritoneal coat, as perityphlitis and inflammation of the retrocæcal (retroperitoneal) cellular tissue, as paratyphlitis.

Inflammation of the cæcum is produced most frequently by fæcal stasis (coprostasis). The fæces act as an irritant and give rise to inflammation. More rarely this is the result of the ingestion of sharp foreign bodies, or of ulcerative changes in the mucous membrane.

Fæcal stasis is more apt to develop in the cæcum, because the small intestine enters it almost at a right angle, and because in this part the fæces first undergo a marked increase in consistence. In addition, the fæces must pass directly upwards along the ascending colon. The danger becomes greater if the muscular coat of the intestine is enfeebled by previous catarrhal or other disease of the cæcum. In many cases, also, we notice errors of diet and irrational habits of life. Individuals who pursue sedentary occupations are predisposed to stasis of fæces in the cæcum. Injury to the right iliac fossa sometimes seems to produce the disease by giving rise to a paralytic condition of the muscular coat of the intestines.

Inflammation of the vermiform appendix is due to the same causes as inflammation of the cæcum.

The retention of foreign bodies and fæces in the vermiform appendix may be aided by its abnormal anatomical structure, or by previous disease. The entrance to the appendix is surrounded by a valve-like projection, the excessive development of which may prevent the exit of substances which have entered it. Biermer and Bossard found that catarrhal inflammation of the vermiform process is not infrequent. If

this affects the muscular coat, it interferes with the discharge of its contents and favors stasis.

The pieces of fæces contained in the appendix are sometimes extremely hard (enteroliths). Enteroliths are divided into two classes, true and false, according as we have to deal with very hard masses of fæces or with earthy deposits (carbonate and phosphate of lime and magnesia). True enteroliths are often laminated, and not infrequently contain a foreign body. They should not be mistaken for gallstones, which may also enter the cæcum and vermiform appendix. Enteroliths are usually round or elongated in shape. The following are the results of chemical analysis by Aberle and Schuberg:

	Aberle.	Schuberg.
Water,	22	57.3
Ammonia magnesia phosphate,	4.3	24.4
Calcium phosphate,	60.5	6.7
Calcium sulphate,	1.1	1.3
Alcohol-ether extract,	0.3	0.8
Other organic substances,	11.3	9.2

It may finally be mentioned that typhoid, phthisical, and catarrhal ulcers may be restricted to the vermiform appendix.

As a rule, paratyphlitis is a secondary disease. The primary form has been attributed to cold, and I have seen a number of cases which resulted from violent bodily exertion. According to Kraussold, walking the tight rope is a frequent cause of perityphlitis and paratyphlitis in children. Secondary paratyphlitis generally follows inflammation of the cæcum; but the inflammation may extend from more remote parts, since the cellular tissue extends to the kidneys, into the pelvis, the abdominal wall, and inguinal ring. Thus, paranephritis and parametritis may give rise to paratyphlitis. The latter may also be secondary to psoriasis, diseases of the pelvic bones, and even to spinal caries. In some cases the disease is a metastatic inflammation. This is observed in pyæmia and puerperal fever, more rarely in certain infectious diseases, such as typhoid fever, articular rheumatism, and measles. Perityphlitis is likewise secondary to inflammation of the cæcum and vermiform appendix in the majority of cases. The disease rarely extends from adjacent organs, for example, in oöphoritis, salpingitis, and perimetritis. Primary perityphlitis after cold or injury does not often occur.

These forms of inflammation are often associated with one another. They are observed much more frequently in men than in women, and usually from the ages of fifteen to twenty-five years.

II. SYMPTOMS.—Typhlitis, paratyphlitis, and perityphlitis give rise to pain and swelling in the right iliac fossa, and to disturbances of digestion.

Typhlitis—we choose typhlitis stercoralis as an illustration—sometimes begins slowly, sometimes suddenly. In the former event, it is sometimes preceded for days by signs of disturbed digestion, constipation alternating with diarrhœa, cutting pains in the right iliac region, eructations, nausea, and loss of appetite. If the disease begins suddenly, the immediate causes can often not be demonstrated.

The patients complain of intolerable pain in the right iliac region, which is increased on the slightest movement or pressure. In order to relieve the tension of the abdominal walls and to diminish the violence of the pain, the patient assumes a passive position of the body (upon the right side, with the body bent forwards and the right thigh drawn upwards).

The fever sometimes exceeds 39° C., the pulse is correspondingly accelerated, usually small and hard. The features express pain, and, after the disease has lasted for a few days, the eyes are sunken; the tongue may be clean or coated; speech is often whispering, as in peritonitis.

The patients often suffer from annoying singultus; vomiting occurs in many cases, sometimes of the contents of the stomach, sometimes of biliary matter, sometimes of masses which have a faecal appearance and odor. Such cases are especially grave, since they indicate impermeability of the intestines. While anorexia is complete, thirst is usually increased. Sleep is almost always restless and disturbed.

The right iliac region is generally more prominent than the left, and not infrequently forms a distinct tumor beneath the abdominal walls. Palpation reveals an increased feeling of resistance, and usually a more or less distinctly circumscribed tumor. This is generally elongated, extends diagonally upwards above Poupart's ligament, is sometimes smooth, sometimes nodular, and may occasionally be indented. It is very sensitive on pressure. There is dulness on percussion over the tumor.

The abdomen is often tympanitic, and the diaphragm is pushed upwards. The upper border of the liver is unusually high, and the apex of the heart is situated in the third and even in the fourth intercostal space, often to the outside of the right nipple line. The urine is generally scanty and concentrated. It contains an increased amount of indican (vide page 80). The bowels are constipated, or a few thin passages first occur, and then obstinate constipation follows.

Recovery often occurs with great rapidity if the masses of faeces can be removed from the intestines. If this does not happen, ulcerative processes develop in the caecum, and may give rise to paratyphlitis or perityphlitis, more rarely to perforation-peritonitis. Death may also occur from ileus or from rupture of the intestines, as the result of excessive distention by faecal masses and gas.

Ulceration of the caecum may be followed by the formation of cicatrices, the retraction of which may give rise to stricture.

Inflammation of the vermiform process produces almost the same symptoms as that of the caecum, but it usually begins more suddenly and violently. The tumor is less extensive, or is altogether absent. Vomiting of faeces is not observed in uncomplicated cases, and meteorism is usually absent. The percussion sound over the iliac fossa remains tympanitic. The disease gives rise to perityphlitis more frequently than to paratyphlitis. Not infrequently we find perforation of the appendix and signs of perforation-peritonitis. The perforation is sometimes prevented for a time by peritonitic adhesions to the omentum or adjacent loops of intestines. The destruction of the vermiform process is sometimes annular; indeed, the outer part may be entirely separated and fall into the abdominal cavity. The inflammation producing foreign body then enters the peritoneal cavity, so that it is often difficult on autopsy to discover the corpus delicti. In some cases the ulcers cicatrize. If they are very numerous, the entire organ may obliterate. If only a few are present, occlusion may occur only at the entrance of the appendix. This may result in the gradual accumulation of a serous fluid, which may distend the organ to the size of a fist.

In paratyphlitis, we find a very painful tumor in the right iliac fossa which appears to lie deep down, because it is surrounded by air-containing loops of intestines. On gentle percussion over the region of the caecum a tympanitic note is heard. Dulness is only produced when the

finger is pressed in deeply. The pains often radiate into the back, shoulders, right hand, or leg. The right testicle is sometimes drawn spasmodically upwards, and some patients complain of dysuria. At times obstinate erections are observed. The patients complain not infrequently of formication and coldness of the right leg, and weakness of this limb sometimes remains long after the other symptoms have disappeared. This is evidently the result of pressure of the exudation upon the nerves of the lower extremity. Edema of the right leg must be explained in a similar manner.

In the most favorable event, the exudation is entirely absorbed. Under less favorable circumstances, the inflammation extends to the pararenal connective tissue, to the cellular tissue of the pelvis, rectum, or groin, and the pus finally perforates externally, or into the bladder, uterus, rectum, or vagina, or appears beneath the groin. The pus may also break through the abdominal walls, the perforation being preceded occasionally by erysipelas, or extensive gangrene of the skin. If there is a communication at the same time with the cæcum, an intestinal fistula is produced. Cases have been reported in which the pus perforated the pleural or pericardial cavities. In some of these cases the paratyphlitis subsides, and the sequelæ alone remain.

In some cases the abscess ruptures into the cæcum, colon, or abdominal cavity. This is followed by symptoms of gangrene and pyæmia, or perforation-peritonitis.

In perityphlitis, the peritonitic symptoms predominate. The tumor in the right iliac fossa is extremely sensitive, and very superficial. Gentle percussion reveals dulness, but this is mixed with a tympanitic sound, on account of the subjacent colon and cæcum. Gerhardt noticed peritonitic friction murmurs (Beath-Bright friction murmur) on auscultation and percussion. The dangers include rupture of the pus externally, or into internal organs, erosion of the vessels and internal hemorrhage, thrombosis of the mesenteric veins with subsequent pyephlebitis, hepatic abscess, pyæmia. Encapsulated exudations in the right iliac fossa sometimes persist for months, even for years, and on slight provocation may suddenly give rise to an acute exacerbation.

The duration of typhlitis, paratyphlitis, and perityphlitis is extremely variable. Typhlitis stercoralis not infrequently terminates in a few days, while paratyphlitis and perityphlitis may extend over months. Improvement is sometimes followed by unexpected relapse, perhaps from very trifling causes, such as incautious movement of the body, straining at stool, etc. The disease exhibits a marked tendency to relapse. In one patient I observed the symptoms of perityphlitis five times within one and a half years.

III. DIAGNOSIS.—These three affections often pass into one another and their differential diagnosis may be impossible.

They should be distinguished from the following conditions: *a.* Simple coprostasis in the cæcum in which inflammatory symptoms are absent. *b.* Intestinal cancer. This affects older individuals and runs a slow, chronic course. *c.* Invagination. Muco-bloody evacuations are here observed. *d.* Floating kidney, when situated in the right iliac fossa. This is determined by the shape of the kidney, its mobility, and sometimes by the perceptible pulsation of the renal artery. *e.* Biliary and renal colic. Jaundice or hæmaturia is present, the pains are intermittent and situated higher up. *f.* Cold abscess in caries of the spine and pelvic bones. Changes are found in the spinal column or the pelvis.

g. Psoriasis. Disturbances of digestion are absent. *h.* Tuberculosis and cancer of the mesenteric and retro-peritoneal glands. Nodular and movable tumors are found.

IV. PROGNOSIS.—The prognosis in inflammation of the cæcum is much more favorable than in that of the vermiform process, paratyphlitis, and perityphlitis. The three latter affections always present a very grave prognosis, and we must be prepared to find that the disease runs a very slow course.

V. TREATMENT.—Prophylactic measures must be directed towards securing regular evacuations from the bowels, especially in individuals who have already suffered from these diseases.

In typhlitis stercoralis, we should employ intestinal infusion in order to remove the contents of the gut. At first the infusion should be repeated every hour until a copious discharge of fæces results. As much water should be injected each time as the intestines are capable of holding. Even after copious evacuations, a tumor not infrequently remains for a time in the right iliac fossa, dependent in part upon an inflammatory, serous infiltration of the intestinal walls.

We place less reliance on the internal use of laxatives, especially in cases which have lasted for some time, because intestinal peristalsis is thereby made excessive. This may prove injurious in advanced inflammation.

In paratyphlitis and perityphlitis, and in inflammation of the vermiform process, we should order absolute rest, fluid food, and give large doses of opium (0.03 every hour or two) until the tenderness is distinctly less; in addition, light warm poultices to the right iliac fossa. If the opium produces constipation, the bowels should be evacuated every other day by means of intestinal infusion. If a circumscribed and fluctuating tumor is demonstrable, it should be opened and treated according to surgical principles. Willard Parker recommended the fifth to twelfth day of the disease as the most suitable for operation. If the infiltration is diffuse and not fluctuating, an incision should not be made. We should then endeavor to produce resolution by warm poultices, iodine applications, potassium iodide, iodoform (gr. 45, vaseline $\frac{3}{4}$ iss.) ointment, or blue ointment. The inunction should be made very gently, in order to avoid starting up fresh inflammation. It is better to apply the ointment constantly to the part by means of a strip of flannel. Repeated blisters have also been recommended.

4. *Stenosis and Occlusion of the Intestines. Enterostenosis and Ileus.*

I. ETIOLOGY.—The symptoms of intestinal occlusion are sometimes preceded by those of stenosis.

The causes of both conditions depend upon changes in the intestinal contents, the intestinal walls, or adjacent organs. Occasionally the symptoms are produced by simple stasis of fæces (coprostasis). This may occur in habitual constipation, or may develop acutely after errors in diet, for example, after an excessive meal of grapes, etc.

In some cases, the disease results from foreign bodies in the intestines, particularly gall-stones and enteroliths. Whether occlusion may be produced by coils of round worms has, to say the least, not been positively proven. Occlusion by gall-stones is more frequent than is generally believed. The obstruction is sometimes the result of the size of the stone, sometimes of its unfavorable position. Calculi which have

passed through the biliary passages will not necessarily pass unimpeded through the much wider intestinal canal. In the first place, the biliary passages are capable of very marked dilatation, and, in addition, a large stone may pass directly into the intestines, through a fistula between the gall-bladder and colon, more rarely between the gall-bladder and duodenum. Finally, several gall-stones may be united into a large mass by means of the fæces.

Enteroliths may attain a weight of four pounds and a diameter of twenty-three cm. In one case, Niemeyer found thirty-two stones, which weighed two and a half pounds. Friedlaender described a case in which the foreign body, which had given rise to intestinal occlusion, was composed of shellac, and similar concretions were found in the stomach. Their entire weight was thirty-two ounces. The patient was a drunkard, and had been in the habit of drinking an alcoholic solution of shellac.

Foreign bodies which have passed through the anus into the rectum, may also give rise to intestinal occlusion.

Changes in the intestinal walls which give rise to stenosis or occlusion may be the result of structural changes, disturbances of innervation or displacement (torsion, invagination, internal and external hernia). Among the structural changes may be mentioned neoplasms which either proliferate into the lumen of the canal or interfere with the dilatability of the intestinal wall. Even hemorrhoids, particularly internal ones, may give rise to symptoms of intestinal stenosis. Cicatrices are not infrequently the cause of stricture of the intestines. This is particularly true of dysenteric cicatrices, while it is extremely rare after typhoid ulcers. A similar condition may also follow catarrhal, follicular, and tubercular ulcers. In the rectum, it is occasionally observed as the result of syphilis.

Disturbances of innervation of the intestinal wall, followed by symptoms of stenosis or occlusion, are sometimes central, sometimes peripheral in their origin.

Obstinate constipation, followed by enterostenosis and ileus, occurs occasionally in diseases of the brain and spinal cord. Symptoms of ileus, which can only be explained by partial paralysis of the muscular coat of the intestines, have been observed after injury of the abdomen, the reposition of external hernia, and in peritonitis. Among the dislocations of the intestines, the most important is invagination or intussusception. This is the most frequent cause of ileus in childhood, and about one-fourth of the cases occur between the third and twelfth months. The lumen of the intestine is not infrequently occluded by torsion upon its own long axis or around the axis of its mesentery, or by the knotting of one loop of intestine around another loop, thus interfering with the lumen of the latter. In addition, a loop of intestine sometimes enters an opening or abnormal fissure, where it becomes incarcerated and impermeable (internal hernia).

This is observed in abnormal fissures in the omentum, suspensory ligament of the liver, round ligament of the uterus, fissures in the bladder or uterus, perforation of the intestine itself, operative injury of the peritoneum.

In this category are also included so-called internal hernia in the strict sense of the term (*hernia diaphragmatica*, *duodeno-jejunalis*, *retroperitonealis anterior*, *cæcales*, *iliaco-subfascialis*, etc.). External herniæ also give rise to occlusion of the intestines as soon as they become incarcerated.

Dislocation of the intestine sometimes occurs as the result of abnormal torsion in consequence of previous peritonitic adhesions. Abnormal ligaments may also act in the same way, most frequently after peritonitis. A loop of intestine sometimes slips beneath a ligament, and is constricted, sometimes the ligament first forms a loop into which the intestine falls; finally, a free ligament may surround the intestines like a bandage. In the latter way, intestinal occlusion is sometimes produced by the normal vermiform appendix, or by diverticula of the intestines. Finally, intestinal occlusion is produced by diseases of adjacent organs, such as tumors of the pelvis, uterus, ovaries, bladder, and other abdominal viscera, by peritonitic exudations, and movable abdominal organs. A loop of intestines, which is distended by feces, sometimes presses so strongly upon an adjacent loop that the latter becomes occluded. This may also be the result of a very fat mesentery. Uterine pessaries have also been known to give rise to occlusion of the intestine.

Intestinal stenosis and occlusion occur at every age, but the period of life exercises a certain influence on the character of the obstruction. In children, we have to deal chiefly with invagination, or at the most, with stenosis from torsion on the axis, or intestinal polypi, while certain other forms, for example, occlusion by gall stones, have never been observed in childhood. Coprostasis plays a prominent part at an advanced age. As a general thing, men are more frequently affected than women, and the disease is more often found among the laboring classes than in those who pursue sedentary occupations. According to Lingen, torsion of the axis of the intestine is frequent in St. Petersburg. He explains this on the ground that the Russians have an especially long intestine, on account of the abundant ingestion of vegetables. According to Leichtenstern, there is one case of intestinal occlusion among three hundred to five hundred cases of death from all causes. According to Fagge, the percentage is 1.4.

II. ANATOMICAL CHANGES.—If the lumen of the intestines is interrupted at any point, the part above the site of occlusion becomes distended from the accumulation of intestinal contents, and is also unusually convoluted, while the portion beneath the site of occlusion is empty and contracted. If the obstruction is situated in the duodenum, the stomach and oesophagus may take part in the dilatation. Immediately above the occlusion, we find firm or semi-solid masses; higher up, fluid and gas. The intestinal walls may be so tense as to rupture.

If the occlusion occur acutely, the intestinal walls appear transparent, thin, and pale; if the changes are chronic, the muscular coat of the upper portions of the intestine are hyperplastic. This is a compensatory process which develops because the passage of feces through the narrowed part requires unusual force. If the power of the muscular coat is paralyzed, there is danger of complete occlusion as the result of fecal stasis. Necrotic and ulcerative changes may be found in the intestinal mucous membrane above the stenosis, as the result of mechanical irritation by firm scybala. Bloody suffusion of the mucous membrane and serous layer is often found in the immediate vicinity of the site of occlusion.

Signs of circumscribed or diffuse peritonitis are often present. Perforation-peritonitis, furthermore, is not infrequent, and renders it very difficult to unravel the mechanical processes connected with the occlusion.

Among the changes in the other organs we may mention the frequent

occurrence of foreign-body pneumonia, as the result of entrance of vomited matters into the air passages. The organs are often extremely dry, as the result of the abundant losses of water in the vomited matters.

In invagination (intussusception), one portion of the intestine is found shoved into another. Two varieties of intestinal invagination are recognized—the agonal and vital. The former presents no clinical interest. It is observed most frequently in children who have suffered from intestinal catarrh and cerebral diseases. It is generally multiple, can usually be replaced with readiness by pulling upon both ends of the intestines, and inflammatory symptoms are entirely absent.

This form is probably owing to the fact that towards the end of life the various sections of the intestines die with varying rapidity, so that a portion which is still in active motion can readily force itself into an adjacent motionless one which has already perished. It is almost always observed in the small intestine, and may be ascending or descending.

Vital invagination is almost always descending. Indeed, the occurrence of ascending invagination has been altogether denied, but Jones describes a case in which the descending colon passed into the transverse colon.

At the point of transition of the entering portion into the emerging portion of the intestine, is formed the lower or inner angle of flexion, while the transition of the emerging portion and of the sheath (enveloping portion of the intestine) is known as the upper or outer angle of flexion. This forms the so-called neck of the invagination. It is readily understood that not only the intestine, but also the mesentery is invaginated. The latter is compressed by the sheath and thus exercises a certain traction upon the invaginated intestine, so that the latter is curved within the sheath, with its concavity towards the mesentery. The invagination acts injuriously in two ways: by narrowing or occluding the lumen of the intestine, and by producing circulatory disturbances in its walls. Thus the invaginated portion may become necrotic, and be evacuated with the fæces. This sometimes results in natural recovery. In other cases it is followed by dangerous hemorrhage; or perforation occurs, because the peritonitic adhesions at the neck of the invagination are not sufficiently firm; or a portion of the intestine is cast off beyond the region of intussusception. Finally stenosis may again develop on account of increasing cicatricial contraction at the site of lesion.

In incomplete intestinal invagination, only a small portion of the circumference of the intestine is implicated.

Invagination has a tendency to increase. For if stasis of fæces develops above the site of occlusion, the weight of the fæces will favor an increase of the invagination. The increase always takes place at the expense of the sheath. This is rolled inwards to a greater and greater extent so that the parts which are situated next to the neck are converted into emergent intestine, and the outer angle of flexion is moved downwards. The invaginated portion of the intestine sometimes protrudes from the anus. Cases have been reported in which the prolapsed portion was one-half metre in length. Multiple invagination is very rare. A double or even a triple invagination is sometimes found at the site of the lesion. It is occasionally found that a portion of the intestine forces itself from below between the sheath and outer part of the invaginated gut, so that there is, to a certain extent, a combination of descending and ascending invagination. The following table is furnished by Leichtenstern:

479 invaginations :

Ileo-cæcal invaginations,	212	(44 per cent)
Ileum invaginations,	142	(30 ")
Colon invaginations,	86	(18 ")
Ileo-colon invaginations,	39	(8 ")

479

Occlusion of the intestine by twisting on its axis around the mesentery develops most frequently at the sigmoid flexure, because the mesentery is here extremely narrow and at the same time very long and movable. Much more rarely we find twisting of the axis of the entire small intestine with the exception of the duodenum or a few loops of intestines.

Among seventy-six cases of torsion of the axis Leichtenstern found

At the sigmoid flexure,	45 cases	(59 per cent)
At individual loops of the ileum,	23 "	(30 ")
At the entire small intestine,	8 "	(11 ")

Occlusion of the intestine, by the development of a knot between the loops, is found most frequently at the sigmoid flexure which twists around the loops of the ileum. Torsion on its own axis occurs most frequently in the ascending colon.

III. SYMPTOMS.—The chief symptoms in stenosis and occlusion of the intestines are disturbances in the passages from the bowels; in the former they are impeded, in the latter they cease entirely. In many cases the sole symptom of stenosis is unusual constipation. Even this may be absent, if the patients take only easily digested food and eschew vegetables, bread, and other feculent articles of food. Any error of diet may give rise to great danger and convert the stenosis into complete occlusion.

Some cases are associated with chronic diarrhœa. This is observed not infrequently in syphilitic or cancerous stricture of the rectum.

The fæces not infrequently assume a peculiar shape, particularly when the stenosis is situated at the end of the large intestine. They are flat, ribbon-shaped, or broken off short, or resemble the fæces of goats or sheep. A furrow may be found in one part, particularly in cases of polypi. It should be remembered, however, that fæces of this character are observed occasionally in other conditions, particularly in inanition.

In some cases, constipation is associated with pain, the result of mechanical irritation of the site of stenosis by the fæces. This is especially true of stenosis of the large intestine. If the lesion is situated at the outlet of the rectum, the act of defecation is apt to be attended with great pain. Hemorrhoids often develop in such cases as the result of venous stasis in the hemorrhoidal veins.

On examination of the abdomen, we sometimes find it markedly distended as the result of accumulation of fæces and gas in the intestines. Vigorous peristalsis is not infrequently visible, and the intestinal movements are often accompanied by borborygmus. The fæces which are accumulated above the stenosis can often be felt through the abdominal walls. The obstruction itself is sometimes felt as a prominence, hardness, or circumscribed site of tenderness.

Examination through the rectum and vagina should never be omitted.

Stenosis of the rectum is often felt directly on palpation, or by means of the sound. Sometimes, however, stenosis is simulated from the fact that the tip of the sound catches in a fold of mucous membrane or in the promontory of the sacrum. Less reliable results are obtained by examination with the speculum or intestinal infusion. The capacity of the rectum is so variable that no reliable conclusions can be drawn from the amount of water which may be injected. In some cases it is advisable to examine the rectum by the introduction of the entire hand and forearm. This requires the administration of chloroform, and a careful introduction of the hand, in order to prevent rupture of the rectum.

The combined method of examination may be employed in resorting to vaginal examination, the fingers of the outer hand being placed upon the abdominal walls.

We have already remarked that the signs of stenosis sometimes pass suddenly into those of intestinal occlusion (ileus). There are three prominent symptoms of ileus, viz., the absence of evacuation from the bowels, or of the passage of flatus, and vomiting of fæces.

It is readily understood that defecation does not occur in occlusion of the intestines. Evacuation of fæces, occasionally even diarrhœal in character, may be observed in the beginning, before the intestinal contents, which are situated below the site of occlusion, have been entirely evacuated. For this reason we must be cautious in giving a prognosis, even if the enema produces an evacuation of fæces after the previous absence of defecation. But if flatus is again discharged, we can usually feel certain that the intestine is again permeable, unless considerable air has been pumped into the lower part of the intestine during the introduction of the enema. Vomiting is an almost constant symptom of intestinal occlusion. The vomited matter consists at first of the contents of the stomach, and finally assumes a fæcaloid or fæcal character. On microscopical examination of the vomited matter, we find débris of food, granular detritus, desquamated epithelium cells; according to Betz, the greenish vomited matter contains algæ. It is erroneously held by some that fæcal vomiting occurs only in occlusion of the large intestine, but this can be readily disproved.

Fæcal vomiting sometimes ceases after the disease has lasted for a long time, and the vomited matters then consist of rice-water like masses mixed with flocculi; this is evidently owing to the fact that all the fæcal masses situated above the occlusion have been removed. Towards the end of life, vomiting sometimes ceases entirely and gives way to singultus.

The objective abdominal changes are similar to those described in stenosis, but as a rule they are more marked. The palpation of any tumor which may be present is accompanied by pain. In addition, we often notice colicky pains which may be associated with vigorous peristaltic movements.

Diuresis is very scanty and occasionally almost entirely abolished. Great importance must be attached to the amount of indican in the urine. It is increased in occlusion of the small intestines, unchanged in occlusion of the large intestines (in the absence of peritonitis which in itself increases the amount of indican). This is owing to the fact that indican is derived chiefly from indol, which forms during the pancreatic digestion of the albuminoids in the intestine and passes off in great part with the fæces. If the small intestine is impermeable, almost all of the

indol is absorbed by the blood, and the indican is then excreted in the urine (*vide* page 80).

As a rule, the severity of the disease is soon shown in the general condition. Collapse occurs with great rapidity, the face becomes cool, the eyes are sunken and surrounded with blue rings, the nose becomes peaked, the patients exhibit a peculiar restlessness, the temperature may be normal or irregularly increased, sometimes almost subnormal. The pulse is usually small but regular, more often accelerated than slowed. If the vomiting is very profuse, choleric form symptoms develop. The skin loses its roundness, the extremities feel as cold as ice, even cramps in the calves may be produced. The sensorium is often unclouded until the end of life. A fæcal odor is sometimes emitted from the mouth of the patient.

Death sometimes occurs in a very short time (to a certain extent, as in cases of shock), probably as the result of anæmia of the brain. In rarer cases, the disease lasts for several weeks, until death occurs from exhaustion. Death may also be the result of suffocation, if the excessive accumulation of gas in the intestines pushes upwards the diaphragm, lungs, and heart to an extreme degree. In a third series of cases, intercurrent accidents terminate the scene; for example, rupture of the intestine above the stenosis and perforation-peritonitis or general peritonitis starting at the site of stenosis. If the loop of intestine becomes adherent to the abdominal walls, perforation may occur through the latter with the formation of an artificial anus. Two loops of intestines sometimes become adherent, and communicate with one another by means of a fistula, and in this way restore the permeability of the intestinal canal. A fistula sometimes forms between the intestine and uterus, bladder, etc. Finally, intestinal occlusion may be attended with the symptoms of pyæmia. The gut ruptures into an encapsulated peritonitic exudation (fæcal abscess); through the agency of the mesenteric veins this gives rise to metastatic abscesses in the liver, occasionally to emboli in the lungs.

Recovery is not frequent. It may occur spontaneously or as the result of treatment. It is recognized by the reappearance of flatus and defecation, the cessation of vomiting, and gradual restoration of strength. At the same time tumors, concretions, foreign bodies, and unusually large fæcal masses are sometimes found in the stool.

The symptoms of intestinal invagination require special consideration. The disease not infrequently begins with a sudden colicky pain in the abdomen. This is soon followed by thin mucous, and particularly bloody stools. We not infrequently feel an elongated, smooth, firmly elastic tumor, which is most frequent near the right iliac fossa, because ileo-cæcal invagination is the most frequent form of the disease. The patency of the anus has often been regarded as a very valuable sign of invagination. Bloody, mucous masses sometimes trickle constantly from the anus. The finger is readily introduced into the rectum, thus showing that we have to deal with paralysis of the sphincter. Not infrequently, however, the patient complains of tenesmus. Hirschsprung attaches importance to the fact that the rectal folds are smooth or drawn upwards. The invaginated part can sometimes be reached by digital examination of the rectum. The originally muco-bloody diarrhœa is very often followed in a short time by symptoms of occlusion, and these prove fatal as in occlusion from other causes. Muscular spasms and convulsions occur not infrequently towards the end of life. A sort of natural recovery occurs

in rare cases from spontaneous exfoliation of the invaginated part of the intestine. Sometimes the exfoliated portion passes off in small shreds, in other cases in the shape of the larger part of the intestine (three metres in length in Cruveilhier's case). Spontaneous exfoliation occurs most frequently from the eleventh to the twenty-first days. It is often preceded by stinking, bloody stools. In the most favorable cases it is followed immediately by recovery. In others, intestinal hemorrhage, rupture or perforation-peritonitis develops, or renewed symptoms of stenosis occur after apparent recovery. The duration of the disease depends upon the degree of stenosis. If the occlusion is complete, life will only be prolonged for a few days, although Kussmaul reports a case in which the symptoms of ileus lasted twenty-three days. In intestinal stenosis, life may be prolonged for months, even for years. Exfoliation of the invagination sometimes does not occur until after the lapse of months. Invagination presents a great tendency to relapse. (In Senator's case a replaced invagination recurred nine times in seventeen days.)

IV. DIAGNOSIS.—With regard to the location of the lesion special attention should be paid to the results of the external and internal examination of the abdomen. Great importance should also be attached to the amount of indican in the urine (increased in occlusion of the small intestine, unchanged in occlusion of the large intestine), but in such cases we should be able to exclude peritonitis, since this in itself may increase the amount of indican in the urine. It is also said that in occlusion of the small intestines the symptoms run a more rapid course. The vomiting and pain are more violent, the meteorism is slighter or entirely absent, the nervous symptoms predominate, and diuresis is diminished or abolished. With regard to the character of the obstruction, the diagnosis is easy if the lesion can be reached from the rectum or vagina, or if we have to deal with incarcerated external hernia. The diagnosis can also be made *post hoc*, if tumors, foreign bodies, etc., appear in the stools, and at the same time the signs of occlusion cease. Invagination is often recognized with facility. It occurs usually in children in whom muco-bloody stools are passed, and the anus is patent. The previous history will enable us to determine whether the occlusion is the result of coprostasis. But we will rarely be able to ascertain with certainty whether an ileus is the result of internal incarceration or volvulus of the intestine.

The signs of ileus (vomiting of fæces, constipation, and absence of flatus) are sometimes observed, although stenosis of the intestine is not found on autopsy.

The symptoms of occlusion may be mistaken for: *a*, the action of certain poisons, for example, arsenic. The diagnosis depends on the history, examination of the vomited matter, and the objective abdominal symptoms. *b*, Cholera. *c*, Peritonitis, in which the causes of the inflammation are only revealed on autopsy.

V. PROGNOSIS.—As a matter of course, this is much more favorable in stenosis than in occlusion of the intestines; in the latter disease it is very grave. The bad prognosis should not, however, lead us to fold our hands and leave the patient to his fate.

VI. TREATMENT.—In the treatment of intestinal stenosis, the chief weight should be attached to dietetic measures. The food should be of such a nature as to be readily digested and absorbed (milk, eggs, bouillon, meat, brandy, beer, wine). In addition, care should be taken

in the mastication of the food. A daily evacuation from the bowels should be secured (vide p. 122).

Certain forms of stenosis may be relieved by operation, for example, in cancer of the rectum and polypi by removal of the obstruction, or in stricture of the rectum by gradual dilatation.

If the signs of occlusion follow coprostasis, as large an amount of water as the intestine will hold should be injected (at first every two hours) until a copious evacuation follows. Then this should be repeated two or three times a day, and in addition cathartics should be given internally. For example: \mathcal{R} Inf. sennæ comp., \mathfrak{z} vi.; Natri sulph., \mathfrak{z} vi. M. D. S. One tablespoonful every two hours. \mathcal{R} Ol. croton., gtt. iij.; Ol. ricini, \mathfrak{z} i.; Gummi arab., \mathfrak{z} ij.; ft. c. aq. destil., q. s., emulsio, \mathfrak{z} vi.; Syr. Sennæ, \mathfrak{z} vi. M. D. S. One tablespoonful every two hours.

The first injections are often ineffective because a certain length of time elapses before the lowermost masses of fæces are softened. Hard masses, which are felt above the anus, can often be removed by the finger. In some cases it is advisable to push the rectal tube through the masses of fæces.

In invagination, the intestine should be quieted as quickly as possible by large doses of opium (gr. ss. every hour in adults until the pupils are distinctly contracted), and then large amounts of water should be injected into the rectum to relieve the invagination. The infusion may be repeated three or four times a day. Distention of the large intestine with gas may also be attempted. This is done most readily by means of a rectal bougie which is connected with the bulb of a Richardson ether spray, care being taken that the bougie is introduced as far as possible into the rectum.

Ziemssen recommended distention of the large intestine by means of carbonic acid gas, which is produced by the combination of tartaric acid and sodium bicarbonate. This required about \mathfrak{z} vi. sodium bicarbonate and \mathfrak{z} v. tartaric acid. These substances, dissolved in water, should be introduced into the rectum in small portions in order to prevent sudden distention.

If the invagination has extended to the lower part of the large intestine, we may attempt to replace it by means of an oiled flexible sound. As relapses occur not infrequently, it may be necessary to retain the sound for some time in the anus. If the invagination cannot be replaced, abdominal section may be resorted to, but the operation only offers chances of success if it is performed as early as possible, and peritonitic adhesions are not present. Among twenty cases of laparotomy in intestinal invagination of children, there were six recoveries. In all forms of intestinal occlusion, the sites of external hernia should be carefully examined, and in cases of incarceration of external hernia, these should be replaced or relieved by operation. If we have reason to assume torsion of the intestine or internal strangulation as the cause of an existing intestinal occlusion, the greatest chances of success are afforded by the operation of laparotomy at as early a period as possible.

Instead of laparotomy, some authors recommend the formation of an artificial anus, enterotomy, above the site of occlusion. This operation seems to be preferable when peritonitis—which interferes very materially, if it does not render impossible, our search for the obstruction—may be assumed to be present. The artificial anus should not be made too large, so that it may be readily closed by an obturator.

Schramm collected the statistics of 199 laparotomies which were performed for intestinal occlusion from various causes, and in which 122 cases proved fatal (61 per cent). Since the introduction of Lister's method of treatment, there were 65 deaths (53 per cent), among 122 laparotomies.

In the majority of cases, however, the cause of the occlusion remains undiscovered during life, or the patients do not consent to an operation. With the aid of the antiseptic mode of treatment, the surgeon is often tempted, even when the cause of occlusion is unknown, to perform laparotomy and to search for the obstruction to the intestine, but the difficulty of the undertaking should not be underestimated. It is usually extremely difficult to unravel the mass of distended and twisted loops of intestines; at all events, laparotomy should not be performed if peritonitic symptoms are present.

When we are restricted to purely internal measures of treatment, we should recommend chiefly the administration of large doses of opium, while the administration of cathartics, which is recommended by some writers, is, in our opinion, very dangerous. Cahn has recently claimed that he repeatedly succeeded in relieving the symptoms of ileus after copious injection of the stomach with water, and thus removing the contents of the stomach and intestines above the stenosis. The stomach should be washed out from three to four times a day. The patient should not be allowed to drink much water, and thirst should be relieved by the ingestion of pieces of ice.

Even in recent times, reliable physicians have recommended the administration of large amounts of mercury (five to ten ounces, even as much as two lbs.), and in desperate cases this may be employed as a *dernier ressort*.

We should not fail to attempt to relieve the obstruction in the intestine by repeated infusions of water and enemata of air into the rectum. I may also mention that in one case in which all methods of treatment had been adopted for almost a week without success, so that the patient was about to be sent to the operating-room, faradization of the intestine produced a copious evacuation from the bowels at the end of three minutes (one pole in the rectum, the other, labile, upon the abdominal walls, particularly near the cæcum and colon); recovery was permanent. In less favorable cases, faradization should be repeated three or four times a day. At all events, in the treatment of ileus no plan of treatment should be left untried.

5. *Round Ulcer of the Duodenum (Ulcus Duodeni Pepticum).*

I. ETIOLOGY.—The round duodenal ulcer has the same origin as the round gastric ulcer, that is, the mucous membrane is digested by the digestive juices, after circulatory disturbances in the tissues. The ulcers are almost always found above the entrance of the ductus choledochus and ductus pancreaticus. This is explained on the ground that the gastric juice which has entered the intestinal canal with the food retains its acid reaction and digestive power, only until it reaches the parts mentioned, while lower down it becomes inactive. In one case, Merkel observed embolism as the cause of the circulatory disturbance. Billroth calls attention to the development of duodenal ulcer in septicæmia, and is inclined to attribute such ulcers to the same cause as the duodenal ulcers, which occur not infrequently after burns of the external integu-

ment. The latter occur usually from the seventh to seventeenth days, occasionally on the second day, after the accident. Very slight burns are sometimes sufficient to produce duodenal ulceration. Congelation is also said to be an occasional cause of duodenal ulcers. Some writers have also associated them with erysipelas, pemphigus, and waxy degeneration of the intestinal vessels. Ulcerative changes of an embolic character are observed even in the new-born, and explain certain cases of melæna neonatorum. The male sex is affected more frequently than the female.

II. ANATOMICAL CHANGES.—In the mucous membrane we find sharply-circumscribed losses of substance, the edges of which are terrace-shaped and free from inflammation. The ulcers are situated generally in the upper horizontal, rarely in the descending part of the duodenum. They sometimes extend from the duodenum to the mucous membrane of the pylorus. They are usually single, more rarely multiple. Peptic ulcers are very rarely found in other parts of the intestine, and then must also be attributed to interference with the circulation which permits the action of the digestive ferments upon the mucous membrane.

Duodenal ulcers are characterized by a tendency to perforation or fatal hemorrhage. The erosion may affect the pancreatico-duodenalis, gastro-epiploica or hepatic arteries, the portal vein and vena cava. Stich described perforation into the aorta, and abnormal communication with the gall-bladder has also been observed. Cicatrization results quite often in stenosis of the duodenum, followed by dilatation of the stomach and œsophagus. Cicatricial occlusion of the ductus choledochus and ductus pancreaticus with chronic jaundice have been observed when the ulcer was situated at the point of entrance of the ducts.

III. SYMPTOMS AND DIAGNOSIS.—The symptoms may be so similar to those of round gastric ulcer that it is hardly possible to differentiate them during life. The most prominent symptoms are pains in the epigastric region, especially on the right side, tenderness of the abdominal walls, vomiting, hæmatemesis and enterorrhagia. The ulcer occasionally develops in a latent manner, and death results quickly after sudden symptoms of perforation-peritonitis, or intestinal hemorrhage. In making a differential diagnosis between round gastric and duodenal ulcers we should notice whether the pain is most marked on the right side, and whether intestinal hemorrhage occurs repeatedly, but hæmatemesis not at all. Some attach great importance to the fact that in round gastric ulcer the pain develops very soon after meals, while not infrequently three to four hours elapse before the occurrence of pain in duodenal ulcers.

IV. The prognosis and treatment are the same as in round gastric ulcer. (Vide p. 73.)

6. *Intestinal Cancer.*

I. ANATOMICAL CHANGES.—Cancer of the intestines is generally primary. It is less frequently secondary to cancer of adjacent organs, extending to the intestinal walls by contiguity; in very rare cases it is metastatic. Primary cancer of the intestine has a tendency to spread in a ring shape, giving rise to stenosis or stricture, which is sometimes so marked that a lead pencil cannot be passed through the narrow portion. In rare cases we find isolated nodules. The cancer may be scirrhus, medullary, or colloid. Colloid is relatively frequent in the rectum, in which pigment cancer (melanocarcinoma) is also observed in rare cases.

The gross appearances depend in great part on the microscopical

structure. Sometimes we find white, dry, and crumbling, or more juicy masses; sometimes a tumor which is infiltrated with a yellowish or brownish colloid fluid; sometimes, finally, the cancer forms a callus-like, firm, almost cartilaginous thickening of the intestinal walls. Melanocarcinoma has a coal-black appearance. The cancerous changes sometimes extend to the muscular coat and serous membrane. The muscular coat is thickened, infiltrated with a meshed connective tissue; it also contains white bands of cancer. If the cancer has given rise to stricture of the intestines, the gut above the stenosis is found dilated and filled with feces and its walls are thickened. Below the stenosis the gut is empty and its walls thin. The intestinal walls may rupture if the tension has been excessive. Cancerous stenosis may disappear on account of partial degeneration of the growth, which passes off in the evacuations. In such places cicatricial tissue may form and, according to Rokitansky, a sort of spontaneous recovery may occur in this way. The degeneration of the tumor is associated not infrequently with considerable danger. Sometimes it gives rise to very violent hemorrhage, or perforation of the intestine occurs, or the adjacent organs become adherent by peritonitic adhesions and perforation then occurs into such organs. In this way the intestine may communicate with the bladder, vagina, and uterus, or, in rare cases, an external fecal fistula forms. Sometimes the perforation occurs into the retroperitoneal cellular tissue, giving rise to a fecal abscess and then to pyæmia.

Primary cancer may be confined to the intestine; in other cases it spreads to adjacent organs, especially those situated in the pelvis, or finally metastases form in the lymphatic glands. Cancer is most frequent in the rectum, particularly at the transition into the sigmoid flexure; next in the flexure itself and the points of flexion of the colon. It is rare in the small intestine, relatively frequent in the duodenum, particularly at the point of entrance of the ductus choledochus or at the origin of the duodenum, into which it usually extends from the pylorus.

Among 34 cases of intestinal cancer (exclusive of cancer of the rectum) collected by Koehler, 22 were situated in the large intestine, and 12 in the small intestine.

II. ETIOLOGY.—Intestinal cancer is more frequent in males than in females, and as a rule, develops after the age of forty years. In rare cases it is observed in childhood. (Wiederhofer reports two cases in infants of three and sixteen days respectively.)

III. SYMPTOMS.—These are sometimes so indefinite that a diagnosis is impossible during life. The patients complain of pain in the abdomen, often in a circumscribed spot. They suffer from irregular evacuations, usually constipation, more rarely diarrhœa. Emaciation and marasmus develop, but the cause of the disease is only discovered on autopsy.

In other cases ileus suddenly develops, errors of diet occasionally being the immediate cause.

In cancer of the lower part of the large intestine, the first symptom is intolerable pain in the sacral region, radiating into the genitals and the distribution of the sciatic nerves. The chief symptoms are demonstration of a tumor and changes in the stools. If the cancerous tumor can be reached through the abdominal walls, it is usually found as a nodular, elongated, roundish tumor. In some cases this attains the size of a fist, or even more. It is generally sensitive on pressure, but, unlike coprostasis, it cannot be indented on pressure. It may be either movable or immovable.

Its mobility sometimes depends upon the part to which it is attached, or upon the development of peritonitic adhesions. When situated in the small intestine or transverse colon, it occasionally presents an extraordinary degree of mobility. Since these parts of the intestine are rendered heavier by the tumor, they often sink very deep in the abdominal cavity. Hence the majority of intestinal cancers are found below the umbilicus. Very great variations are sometimes found, at brief intervals, in the distinctness with which the tumor is felt. Furthermore, in examinations at different times, we often notice a remarkable change in the size of the growth as the result of varying distention and position of other loops of intestine. The tumor gives a dull tympanitic percussion sound, but the pleximeter must be firmly pushed into the abdominal walls.

Rectal cancer is particularly accessible to examination through the rectum. Sometimes we find an annular, usually smooth stenosis of the rectum, sometimes ulcerated nodulated surfaces. On combined examination (one hand upon the abdominal walls), the changes are often recognized more distinctly. The finger, on its withdrawal, is usually found covered with foul-smelling muco-purulent, and often bloody fluid. Suspicious débris may also be examined under the microscope (vide page 43).

In rare cases, prolapse of the rectum occurs. When the cancer is situated very low down, it may become visible to the eye. In other cases, the rectal speculum may be employed, although this usually gives rise to great pain.

The stools may be changed with regard to amount, shape, and constitution. In the majority of cases there is obstinate constipation. Cooper Foster reported a case of constipation lasting eighty-eight days in colloid cancer of the descending colon. The constipation sometimes ceases quite suddenly, and is followed by frequent evacuations. This must be attributed to ulceration of the cancer and relief of the stenosis, or to the formation of a fistulous communication between the stenotic part of the intestine and lower parts. The fæces sometimes become flattened and tape-like, or are voided in small round lumps like the fæces of a sheep.

Violent intestinal hemorrhage is observed in some cases. In others, the stools contain ichorous, mucous, muco-purulent, or bloody masses, in which particles of cancer are occasionally seen. All other symptoms are unreliable (pain, emaciation, and increasing cachexia).

Special symptoms, dependent upon the situation of a cancer, are observed in not a few cases. In duodenal cancer, which is situated near the papilla of the ductus choledochus, obstinate jaundice is produced, while in cancer of the first part of the duodenum, the signs of stenosis of the pylorus become evident.

In rectal cancer, the general condition remains good for a very long time. The evacuations may be followed by the most violent pains, so that many patients retain the stool as long as possible. Obstinate diarrhoea occurs not infrequently. Thin, ichorous masses, mixed with blood, may trickle constantly from the anus. There is often very marked dilatation of the hæmorrhoidal veins, and indeed we must be on our guard against mistaking the disease for bleeding piles.

The duration of the disease may extend to four years. In one of my cases death occurred at the end of the fifth year. Some patients die from increasing marasmus. Occasionally, secondary cancer in the liver or other organs is the real cause of death. In some cases, the symptoms of intestinal stenosis and ileus give rise to the fatal termination. This may also

result from perforation of the intestine, the development of fæcal abscesses, with pyæmia, or abnormal communication with other organs.

IV. DIAGNOSIS.—In not a few cases the diagnosis is impossible; in many others, extremely difficult. Cancer of the rectum and sigmoid flexure alone is easily recognized if a digital examination of the rectum is performed.

If the tumor can be felt through the abdominal walls, it may be mistaken for fæcal stasis (which does not always disappear after the administration of cathartics, especially if they are not repeated daily for a number of days), tumors of the stomach, pancreas, liver, lymphatic glands, kidneys, omentum, or encapsulated peritonitic exudations, etc. The diagnosis is rendered positive by the discovery of elements of cancer in the stools, but this occurs very rarely.

FIG. 18.



Microscopic appearance of a spontaneously desquamated, intestinal polypus, in a girl æt. 10 years
Enlarged 275 times.

V. PROGNOSIS.—The prognosis is bad, although it has been improved by modern surgery.

VI. TREATMENT.—The treatment of intestinal cancer belongs to surgery rather than to internal medicine. Internal remedies are indicated only when secondary deposits in other organs render an operation useless. We should order a nutritious diet, which will give rise to the minimum amount of fæces; milk, eggs, meat, beer, wine, soup. If constipation is present, we may give mild laxatives or enemata; if the pain is violent, subcutaneous injections of morphine, or, in rectal cancer, suppositories of morphine or belladonna. If there is an ichorous discharge from the rectum, the large intestine should be washed daily with an infusion of water, to which is added kalium hypermanganicum (one per cent), or liq. alum. acetic. (3 i. : 3 iij. One teaspoonful to a glass of water).

APPENDIX.

The other neoplasms of the intestines are interesting to the anatomist or surgeon rather than to the physician.

a. Polypi.—We may find pedunculated fibromata, which start from the submucosa, or mucous polypi, dependent chiefly on proliferation of the mucous glands. They are situated most frequently in the rectum, immediately above the sphincter, are single or multiple, and are found particularly in children, in whom they produce diarrhœa and mucopurulent discharges. The polypi sometimes protrude from the anus during evacuation. The fœces sometimes present a furrow, which has been produced by the tumor. The polypi are occasionally separated from the pedicle during defecation, and are then voided, giving rise to slight hemorrhage. Large growths sometimes give rise to intestinal invagination, or to sudden stenosis.

b. Lipomata may give rise to the same symptoms as polypi, but are much rarer.

c. Angiomata, myomata, sarcomata, and cystomata of the intestines have also been observed.

7. *Animal Parasites of the Intestines. Helminthiasis.*

Animal parasites in the intestines are included either among the protozoa or the worms. Among the former, we distinguish rhizopods and infusoria, among the latter flat worms (platodes) and round worms (nematodes).

The following list includes those parasites which possess a practical interest:

First Group. Protozoa.

Rhizopods.

Amœba coli.

Infusoria.

Cercomonas intestinalis.

Trichomonas intestinalis.

Balantidium coli.

Second Group. Worms.

Flat worms. Platodes.

Tœnia solium.

Tœnia saginata.

Bothriocephalus latus.

Round worms. Nematodes.

Ascaris lumbricoides.

Oxyuris vermicularis.

Trichocephalus dispar.

Anchylostomum duodenale.

Trichina spiralis.

PROTOZOA IN THE INTESTINES.

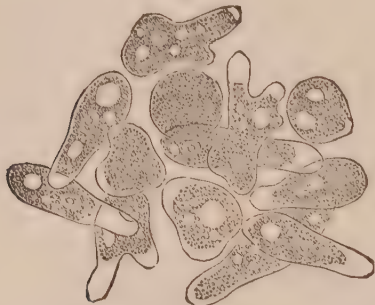
There are hardly any differences in the clinical symptoms produced by the various forms of protozoa in the intestine. They are almost always found in individuals who suffer from chronic diarrhœa, more frequently in typhoid fever patients.

The relation of protozoa to the diarrhœa has not been positively

determined. In some cases, the diarrhœa ceases after the protozoa have been removed from the intestine by the use of enemata, but this may have been the effect of the enemata themselves.

In individuals who suffer from obstinate chronic diarrhœa, the fæces

FIG. 19.



Amœba coli. After Loesch.

FIG. 20.



Cercomonas intestinalis. After Lambl.

should be examined fresh under the microscope, since the parasites become immovable after a time, assume a spherical shape, and thus escape observation.

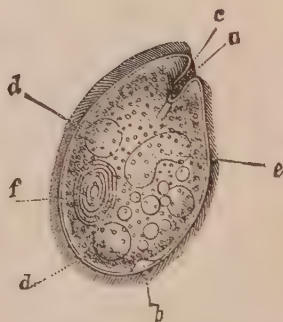
According to Nothnagel, the occurrence of protozoa in the intestines is almost a normal condition.

FIG. 21.



Trichomonas intestinalis. After Zunker.

FIG. 22.



Balantidium s. Paramoecium coli. a, peristoma; b, anus; c, row of cilia; d, contractile vesicles; e, nucleus; f, ingested granules of starch flour.

We will add the chief morphological characteristics of the individual forms:

a. *Amœba Coli*.—This was observed once by Loesch in a Russian peasant, who suffered from chronic dysenteric attacks. The amœba appears in the shape of round, granular structures, 0.02–0.35 mm. in size,

with a nucleus and nucleolus and a number of vacuoles (Fig. 19). It is capable of changing its locality.

b. Cercomonas Intestinalis.—The animal is pear-shaped, with a short prolongation posteriorly; anteriorly is a long whip by means of which locomotion is mainly effected (length of body, 0.008–0.01 mm.). The parasite has been observed in mucous evacuations of children, in the stools of cholera, typhoid fever (Fig. 20). Attention has been called to the foul odor of the stools.

c. Trichomonas Intestinalis.—The animals are almond-shaped, 0.01–0.015 mm. long, and 0.007–0.01 mm. wide (Fig. 21).

The anterior portion of the body possesses a fringe of cilia in active motion. The parasite has been observed in acute and chronic diarrhœa, the stools of typhoid fever, and the tartar of the teeth.

d. Balantidium s. Paramœcium Coli.—This animal has been observed in man only in the vicinity of Stockholm, Upsala, and Dorpat. Leuckhart showed that it is always present in the large intestine of the pig.

The animal is pear-shaped, and 0.07–0.1 mm. in length (vide Fig. 22). The interior is granular, and usually contains two vacuolæ, together with particles of food, débris of plants, red and white blood-globules.

Henschen and Waldenstroem recommended the destruction of the parasite by intestinal infusion of a can of water, $\frac{3}{4}$ iss. vinegar and 3 iss. tannic acid (at a temperature of 37° C.).

FLAT WORMS IN THE INTESTINES.

Platodes. Tape Worms.

I. SYMPTOMS.—Among the various forms of tape-worm, three possess practical importance, viz., *Tænia solium*, *Tænia saginata* (mediocanellata), and *Bothriocephalus latus*. The patients sometimes enjoy the best of health, and the presence of a tape-worm in the intestine only becomes evident after the passage of pieces in the fæces or the accidental discovery of the ova under the microscope.

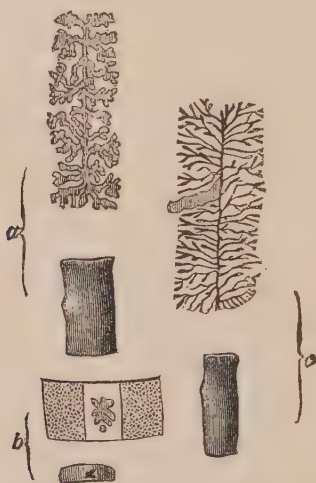
In another series of cases, purely local disturbances of the stomach and intestines are observed, and may last for years before their cause is discovered. Many patients complain of a feeling of pressure in the abdomen which is sometimes constantly present in one place, sometimes passes from one part to another. Colicky pains are observed not infrequently. Some patients state that they experience a peculiar sensation in the intestine, as if a long worm were moving about; but this is probably the result of a preconceived notion. These local symptoms are often produced or increased by the ingestion of certain articles of food. This is particularly true of herring, onions, garlic; while milk, eggs, and oily substances relieve the symptoms. Many patients complain of frequent vomiting, particularly in the morning. Among the frequent symptoms are disturbances of the appetite and digestion. Some patients present an almost insatiable appetite, but continue to emaciate despite the astonishing amount of food ingested. Others suffer from obstinate loss of appetite.

The bowels are sometimes constipated, sometimes diarrhœa is noticed. General symptoms may be superadded to the local disturbances.

Indeed, there is scarcely an organ in which the morbid symptoms have not been observed. As a matter of course, purely accidental complications may be regarded as the reflex symptoms of the presence of the tape-worm. The proof of the connection between the two is afforded only by the immediate disappearance of the symptoms after the removal of the parasite.

Among the general symptoms may be mentioned obstinate singultus, dizziness, pain in the head, syncope, delirium, mania, spasms, chorea, paralysis, difference in the pupils, disturbances of sight and hearing, etc. From time to time, mature links of the tape-worm are spontaneously evacuated in the fæces. This occurs most frequently in spring or autumn, because the tape-worm requires a certain time before its development is completed. Sometimes the tape-worm is cast off in toto. I have repeatedly observed this in children or adults who were subjected to treatment with cathartic mineral waters. Certain articles of diet, which are disagreeable and injurious to the tape-worm, or the administration of

FIG. 23.



Proglottides from *a*, *Tænia solium*; *b*, *Bothriocephalus latus*; *c*, *Tænia saginata*. Lower row natural size; upper row, enlarged 8 times.

laxatives, may also cause their spontaneous evacuation. This is sometimes observed during febrile diseases, particularly typhoid fever. In certain cases, it is the result of an affection of the tape-worm itself, as is often shown by certain malformations of the individual links.

Peroncito found that cysticeri and tape-worms exhibited lively movements at a temperature of 30° to 35° C., which continued up to 48° C. and at 50° C. were permanently lost.

Spontaneous evacuation of pieces of the tape-worm occurs most frequently, though not always, through the anus. In rare cases, they may be vomited.

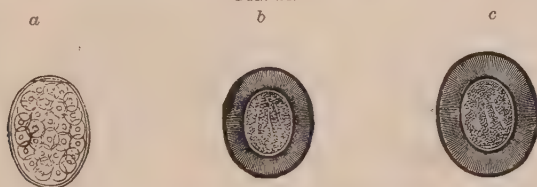
The appearance of links of the tape-worm in the fæces not alone renders the diagnosis certain, but also enables us to determine the variety of tape worm. In *Bothriocephalus*, the individual links are broader than they are long, and at the middle present a dark, often pigmented

spot, corresponding to the sexual opening. In the two varieties of *tænia*, the links are longer than they are broad, and the sexual opening is indicated by a slight prominence to one side. In *tænia solium* the uterus, which is situated in the middle, sends out seven to twelve thicker and less branching lateral shoots, while in *tænia saginata* the ramifications are much more abundant (fifteen to twenty). In *tænia solium* these ramifications are shaped like the branches of a tree, in *tænia saginata* they are simply fork-shaped.

The examination of the members is best effected by slightly compressing them between two object glasses. In examination with transmitted light the sexual parts then appear distinctly; when still alive the parts not infrequently present vermicular contractions. In *Bothriocephalus latus* the links are usually cast off in the shape of small chains, the *tænia* are cast off in single pieces or very few are joined together.

On microscopical examination the difference between the ova of *Bothriocephalus latus* and *Tænia* is easily recognized. The egg of *Bothriocephalus* (vide Fig. 24, *a*) is oval and from 0.06–0.07 mm. in length. It has a brown shell of waxy consistence, on the posterior end of which is a removable cover; the interior of the egg has a cellular structure. The ova of *Tænia solium* and *Tænia saginata* differ from one another

FIG. 24.



Ova of *a*, *Bothriocephalus latus*; *b*, *Tænia solium*; *c*, *Tænia saginata*. Enlarged 300–350 times.

only in size. The former are smaller, being 0.032 mm. in width and 0.036 mm. in length, while the ova of *tænia saginata* are 0.035 mm. wide and 0.039 mm. long. They are oval and have a thick shell composed of radiating rods. Their interior consists of granular protoplasm in which six small hooklets of chitin are visible.

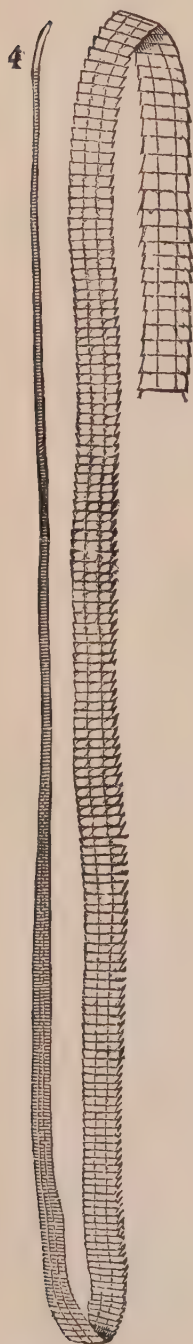
II. ANATOMICAL CHANGES.—Tape worms are located in the small intestine. They adhere to the mucous membrane by means of suction apparatus, in part by means of hooklets located in the head; then there are usually a few loose turns round the neck, while the rest of the body extends towards the large intestine, and occasionally reaches into the colon. Not infrequently the worm is found rolled up into a knotted mass. In the majority of cases only one tape-worm is present, but Berenger-Féraud observed twelve *tæniæ* and Kleefeld forty-one in one individual. As a rule, tape-worms in the intestine belong to the same variety, but both kinds of *tæniæ* may be present, less frequently *tænia* and *bothriocephalus*.

Not infrequently we find other parasites, such as *ascaris*, *oxyuris*, *trichocephalus*, or *anchylostomum*.

a. *Bothriocephalus latus* (vide Fig. 25).

This worm is 5 to 8 metres in length. The number of its links may reach 4,000. The head is club-shaped or almond-shaped, about two mm. in length and one mm. at its widest part. Under the microscope a deep elongated suction-groove is found on each side of the head (vide Fig. 25, *B*). The head is followed by a thread-shaped, thin, cervical portion, 3 to 5 mm. in length. This is followed by the individual members or proglottides.

FIG. 25.

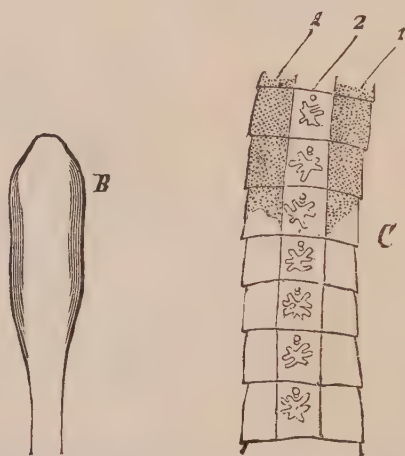


The first ones are considerably broader than they are long, the last have a more quadrilateral shape. The first sexually mature proglottides occur about at the six hundredth link. These are about 5 mm. in width and 2 mm. in length. The quadrilateral ones are about 5 mm. in width and length.

b. Tænia solium (vide Fig. 26). The length of the mature worm varies from two to two and a half metres. The head is round, about as large as the head of a pin, and its tip is not infrequently of a smoky gray or blackish color. Under forty to fifty magnifying powers, we find upon the top of the head a projection (rostellum) which is surrounded by a row of hooklets. The hooklets vary in size, a larger one alternating with a smaller one. To a certain extent two rows are formed, the outer one by smaller,

FIG. 25.

FIG. 25.



Bothriocephalus latus. A, head and anterior portion (natural size); B, enlarged head with the two lateral suction grooves. C, Impregnated links. 1, lateral stripes; 2, middle stripe.

the inner one by the larger hooklets. They vary from twenty-six to fifty in number.

On the sides of the head are four suction-cups, and under the microscope these may be seen to protrude and retract. The head is followed by the thick cervical portion which is 5 to 10 mm. in length. In mature worms, there are about 850 proglottides, of which only 80 to 100 are sexually matured. The latter are 9 to 12 mm. in length, 5 to 10 mm. in width.

c. Tænia saginata (mediocanellata). This attains a length of 7 to 8 metres, and is distinguished from *tænia solium* by the greater development of

the individual links. The head is larger than that of *tænia solium* (about 2.5 mm. wide, in *tænia solium* 1.3 mm. wide), but possesses no rostellum or hooklets. It is also often pigmented at the apex, and has four suction cups. The neck is only 1 to 1.5 mm. in length.

The body is composed of 1,200 to 1,300 links. The sexually mature proglottides begin at about the 600th. The most mature ones have the

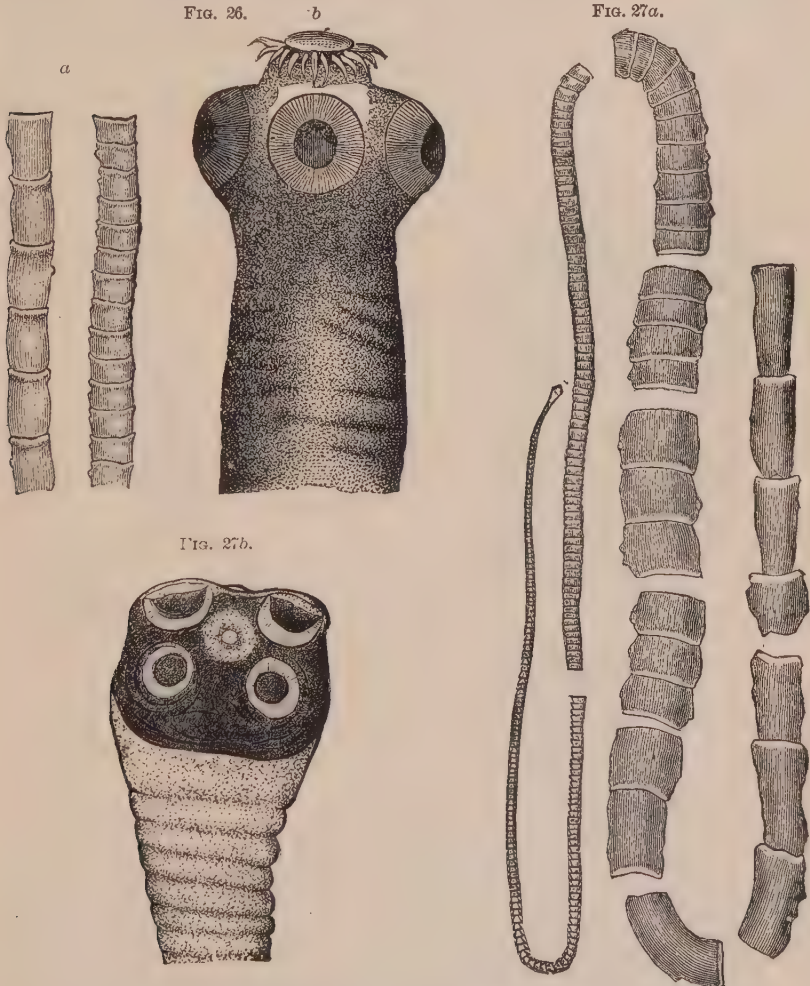


FIG. 26.—*Tænia solium*. *a*, natural size; *b*, head enlarged 450 times. After Leuckart.
FIG. 27.—*Tænia saginata*. *a*, natural size; *b*, head enlarged.

shape of pumpkin seeds, and are 15 to 20 mm. in length, 5 to 7 mm. in width.

Any hemorrhages, erosions, or other lesions which may be found in the intestines must be regarded as accidental complications. According to some statements, a worm may live for thirty years in the individual.

III. ETIOLOGY.—Tape worms are not conveyed directly from one individual to another, because the ova undergo development outside of

the human organism, and must develop into cysticerci before the latter can develop further into tapeworm in the human intestines. Sexually ripe proglottides are cast off by the tapeworm in the human intestine, and either these or the ova alone are devoured by animals. In the latter the ova develop into cysticerci, and these are eaten by human beings in whose intestines they develop into tape worms. *Tænia solium* is developed from the cysticercus cellulosæ of swine, but this is also found in the dog, deer, monkey, bear, and, according to some, in the sheep. *Tænia saginata* develops from the cysticercus of beef; the latter has also been found in the giraffe. The developmental history of *Bothriocephalus latus* is still partly unknown. It is assumed that the eggs enter the water, and then are either swallowed and developed in the in-

FIG. 28.

FIG. 29.

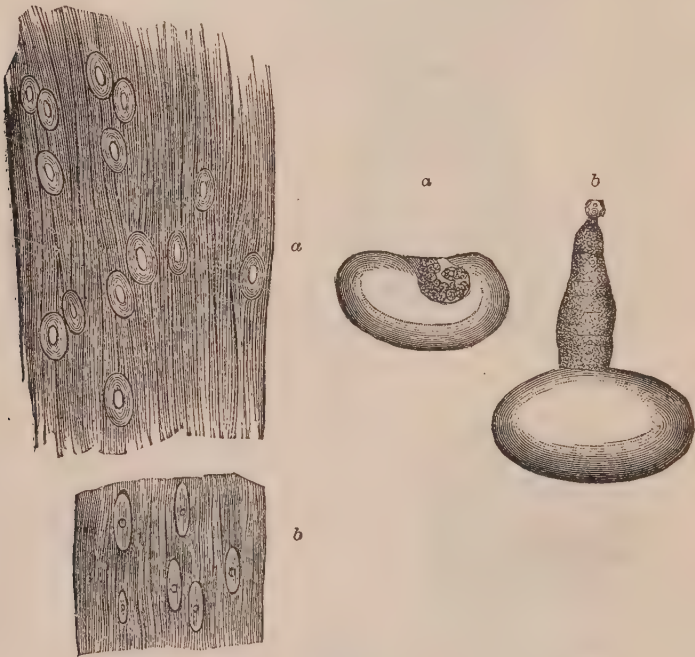


FIG. 28.—Cysticerci. *a*, in pork; *b*, in lamb. After Leuckart.
FIG. 29.—*Cysticercus cellulosa*. *a*, with retracted; *b*, with protruded, head elevation. After Leuckart. Enlarged 1 diameter.

testine into a tape worm, or this occurs after they have previously been partly developed in fishes or aquatic birds. Braun has recently maintained that the scolex of *bothriocephalus latus* is especially frequent in the muscles and intestines of the pike and turbot. It seems, however, that these fishes are particularly apt to be affected when living in the Russian East Sea Provinces, while none are found in those caught in Switzerland. Zaesslein has explained the occurrence of *bothriocephalus* on the shores of the lakes in west Switzerland from the ingestion of lettuce, which grows in ditches which are irrigated by the lakes.

Certain habits exert an influence on the development of tape worms in the human body. Uncleanliness, the defecation of fæces loaded with the ova of tape worm in open places, so that the fæces may be eaten by swine, or the ova may mingle with the drinking water and fodder and

thus be conveyed into the stomach of the animal, which serve as intermediaries; the eating of raw meat, etc., are especially favorable to the spread of tape worms. Individuals are apt to suffer from tape worms in places in which it is customary to eat raw meat; certain occupations also furnish a large contingent; for example, butchers, cooks, etc.

Bothriocephalus latus is found in the western parts of Russia, Poland, East Prussia, Pommerania, Holland, Belgium, North Sweden, Finland, and the western cantons of Switzerland. Zaesslein recently showed that several zones can be distinguished at the Swiss lakes; one at the shore of the lake, in which one person out of five to ten is affected; then a second zone, in which the parasite is found less frequently, finally a zone of immunity, at a distance of four to five miles from the shore.

Tænia saginata is most widely diffused, because beef is everywhere eaten. *Tænia solium* is much rarer. Some races (Jews, Orientals) if they abstain from eating pork, remain free from the worms.

IV. TREATMENT.—Prophylactic treatment is very important. In the first place, the receptacle for fæces should be inclosed, so that the excrement cannot be eaten by pigs. In addition, there should be a compulsory examination of meat by sanitary officers.

We add here two plates of cysticerci in pork and lamb. They are situated in the intermuscular connective tissue, are elongated yellow vesicles 8 to 10 mm. in length, and running in the direction of the muscular fibers. The situation of the so-called head elevation is shown by a bright, usually somewhat retracted and firmer portion which appears in the plates as the clear centre.

Butcher stores should be kept extremely clean. The various kinds of meat should be separated from one another, and a separate knife employed for each. We should caution patients against the ingestion of raw meat. Moreover, the meat should be well done.

Active treatment should not be begun until the presence of the tape-worm is shown by the evacuation of its links in the fæces. If the statements of the patients cannot be trusted, we should attempt to produce evacuation of proglottides by the administration of small doses of laxatives or vermifuges. For example:

℞ Hydrarg. Chlorid. mite,
Jalapæ,
Sacch alb., āā gr. ivss.

M. f. p. One powder morning and evening.

℞ Extract. Filicis,
Rhizomat. Filicis, āā gr. xxx.

Ft. pil. No. xx. D. S. Ten pills to be taken morning and evening.

The treatment proper may be divided into three parts: The object of preparatory treatment is to empty the intestine as thoroughly as possible, in order to facilitate the discharge of the tape-worm. For three days the diet should consist chiefly of milk, soup, and eggs, and several passages a day should be secured by mild laxatives. At night we may give a considerable amount of herring salad, mixed with onions and garlic. Vermifuges proper usually do not kill the parasite, but merely benumb it. Among the numerous remedies of this class, we give the preference to filix mas:

℞ Extract Filicis,
Rhizomat. Filicis, āā gr. 75.

Ft. pil. No. xxx. D. S. Fifteen pills should be taken about 7 A.M., and again at 7:30 A.M.

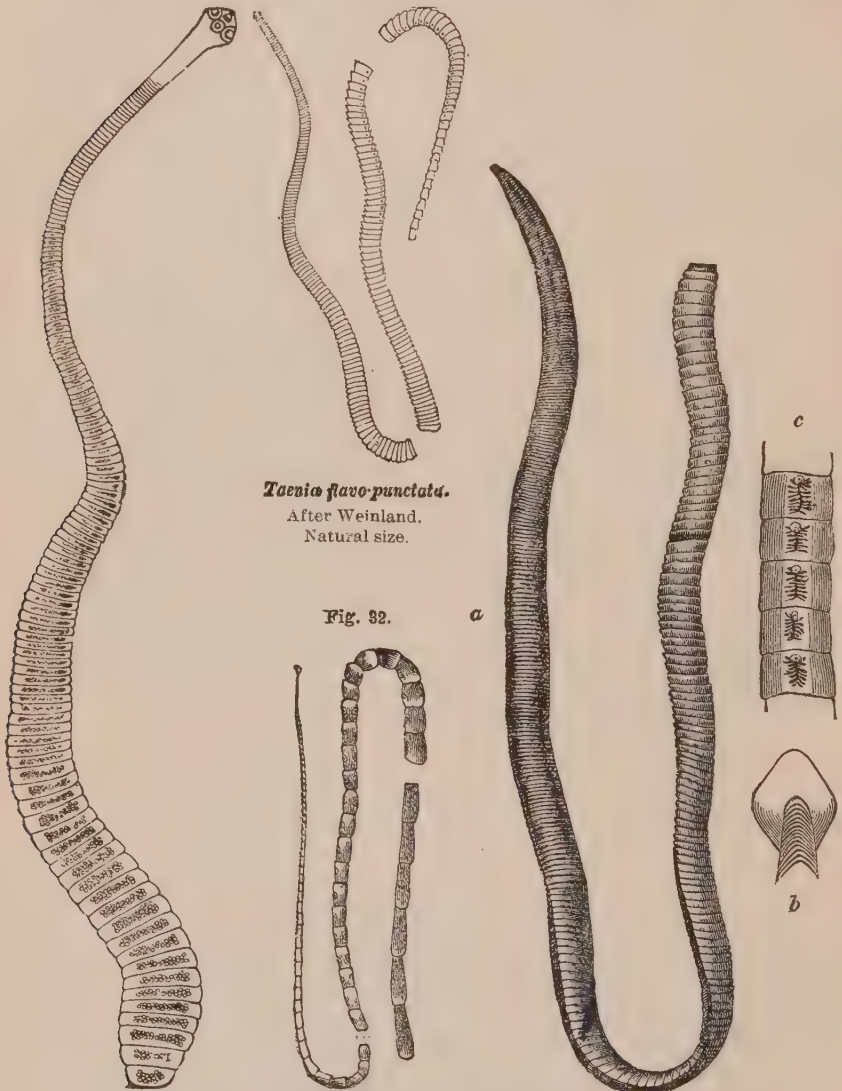
The patient should remain in bed and may take only coffee, or in

case of nausea, lemonade or a little brandy. Sometimes the tape-worm is passed in toto at the end of two to three hours, in other cases it is removed in pieces. We should avoid pulling upon ends which hang

FIG. 30.

FIG. 31.

FIG. 33.



Taenia flavo-punctata.
After Weinland.
Natural size.

FIG. 32.

a

c

b

FIG. 30.—*Taenia nana*. Enlarged 18 times. After Leuckart.
FIG. 31.—*Taenia cucumerina* s. *elliptica*. Natural size. After Leuckart.
FIG. 32.—a, *Bothriocephalus cordatus*. Natural size. After Leuckart. b, Heart-shaped head, enlarged. c, Mature links.

out of the anus, since the tape-worm may thus be torn and the success of the treatment destroyed.

Treatment with cathartics is employed if the tape-worm cannot be evacuated in toto at the end of an hour. It is also useful even after

the worm is removed, in order to clear the intestine as quickly as possible of the remedies which have been administered. Within three hours after taking the pills which have been recommended above, one tablespoonful of castor-oil should be given every hour until the tape-worm is discharged. In addition, an infusion of pure water in the large intestine should be made four times a day. Or if the tape-worm has not made its appearance, we may order : \mathcal{R} Decoct. rhizom. filic., \mathfrak{z} ij. : \mathfrak{z} xx. The treatment can only be regarded as successful if the head of the parasite is discharged. If the worm has been evacuated in pieces, the fæces should be carefully stirred in water and thoroughly examined. If the head is not found, the result remains uncertain ; but the patient may usually be regarded as cured if no new links have been evacuated within ten to fifteen weeks.

We may also mention the following remedies :

a. Flores kousso, \mathfrak{z} vi. to be taken in two portions in the morning, in sugar water, lemonade, or red wine. *b.* Koussinum, \mathfrak{z} i. to be taken in the morning, in two portions. *c.* Camala, \mathfrak{z} ss. to be taken in coffee, in the morning, in two portions. *d.* \mathcal{R} Cort. rad. granat. \mathfrak{z} xiiij., aq. frigida \mathfrak{z} x., macerate for twelve hours, then boil down to \mathfrak{z} viij., add syr. zingib. \mathfrak{z} i. *D. S.* to be taken in the morning in two portions. *e.* Ol. terebinthin., \mathfrak{z} xiiij. in two portions. *f.* Ol. Chaberti or benzolum, 60-200 drops in capsules, etc.

The patient should be kept under observation for some time after treatment. I have seen a number of cases of chronic catarrh of the large intestine and serious exhaustion in which the patients asserted that their disease was the result of treatment adopted against the tape worm.

APPENDIX.

A few examples of other forms of tape-worm have been found in the human intestines. We will mention : *a.* *Tænia nana* in large numbers was observed by Billharz in the duodenum of a child who had died from meningitis. This tape-worm does not exceed 15 mm. in length, has a round head with rostellum, and four suction cups. The rostellum is surrounded by twenty-two to twenty-four hooklets. It has one hundred and fifty to one hundred and seventy links, the last twenty to thirty containing matura ova. (Vide Fig. 30.) *b.* *Tænia flavo-punctata* was observed in one case by Weinland. It attained a length of three hundred mm. The structure of the head is unknown. The anterior half of the tape-worm consists of immature links, each of which contains a yellow patch, while the posterior links present a profuse brownish discoloration, the result of an accumulation of ova (vide Fig. 31). *c.* *Tænia cucumerina* s. *elliptica* has been found a number of times in children. This parasite is the tape-worm of dogs and cats. It attains a length of one hundred and fifty to two hundred mm. The head possesses a rostellum which can be protruded, and beneath which are sixty hooklets, arranged in four irregular rows. The anterior end of the body is very thin. At the posterior end the sexually ripe links again diminish in size. The number of links may exceed one hundred. The color of the sexually mature ones is reddish. (Vide Fig. 32.) *d.* *Tænia madagascariensis* has been observed twice on an island near the coast of Madagascar. *e.* *Bothriocephalus cordatus* was described by Leuckart (vide Fig. 33). It is found, in Greenland, in the small intestine of men and in the dog. It may attain a length of one hundred and fifteen cm., has a short heart-

shaped head, which passes directly into the broad belly. The number of links varies from four hundred to six hundred. A longitudinal furrow is visible on the ventral surface, still more distinctly upon the dorsal surface. The mature proglottides are quadrilateral in shape, with a diameter of from five to six mm. The uterus rosette has from six to eight lateral horns.

ROUND WORMS IN THE INTESTINES. NEMATODES.

Round Worm. Ascaris Lumbricoides.

I. ANATOMICAL CHANGES.—This animal is a round long worm of whitish or pale rose, sometimes brownish-red color. The female is longer (thirty to forty cm.) than the male (twenty cm.). The latter is also more slender and its caudal extremity is usually curved or rolled in towards the ventral surface (vide Fig. 34, B). Transverse furrows in close proximity to one another are readily recognized, and without great difficulty we can also see four longitudinal stripes which run from the head to the tail. Two of these are situated upon the ventral and dorsal surfaces, and the other two run along each side. The head sits like a button upon the rest of the body. At the posterior caudal extremity in the male is found the cloaca, from which the spicula often protrudes. This opening must be looked for in the previously-mentioned ventral stripe (vide Fig. 34, B). The sexual opening in the female is also found in the abdominal line, at the boundary of the anterior and the middle thirds of the body.

A small excretion-pore is found upon the abdominal line, in both sexes, not far from the head. The ova are easily recognized. They have an elongated, rounded shape, and are about 0.05 to 0.06 mm. in length. Their contents are granular, and surrounded by a firm, dark membrane, which in turn possesses an albuminoid covering. The latter presents nodular projections. Leuckart and Eschricht estimate the number of ova in the genital canal of a female at sixty millions. The parasite inhabits the small intestine and is found exceptionally in the large intestine. It is often present in very large numbers.

II. ETIOLOGY.—The developmental history of *Ascaris lumbricoides* is unknown.

Attempts to produce ascaris in the intestines by ingestion of ova have been attended with negative results. It is therefore supposed either that a go-between is necessary, or that worms which have partly developed outside of the human body enter the intestinal tract. Vegetables, fruits, and drinking water have been regarded as the sources of infection. The worm is found very frequently in women and children. It is especially frequent among the insane and in the Orient.

III. SYMPTOMS AND DIAGNOSIS.—The local symptoms include loss of appetite, boulimia, perverse sensations of taste, fœtor ex ore, tenderness of the abdomen, colicky pains, and irregularity of the bowels. The patients, particularly children, grow very pale, change color quickly, and grayish and bluish-gray rings appear around the eyes. Among the general symptoms may be mentioned a tickling sensation in the nose, so that the patient often introduces the finger into the nostrils, irregularity of the pupils, dizziness, syncope, spasms, epilepsy, chorea, meningitic symptoms, paralysis, disturbances of sight and hearing, etc.

Huber also found that ascarides seem to produce an irritation in a chemical (toxic) way, for after handling them he experienced itching

around the head and neck; then wheals made their appearance, the ear became swollen, the external auditory canal began to secrete, conjunctivitis and chemosis developed, together with an annoying throbbing in the head.

The diagnosis may be made either when the worms are passed in the

FIG. 34.

A

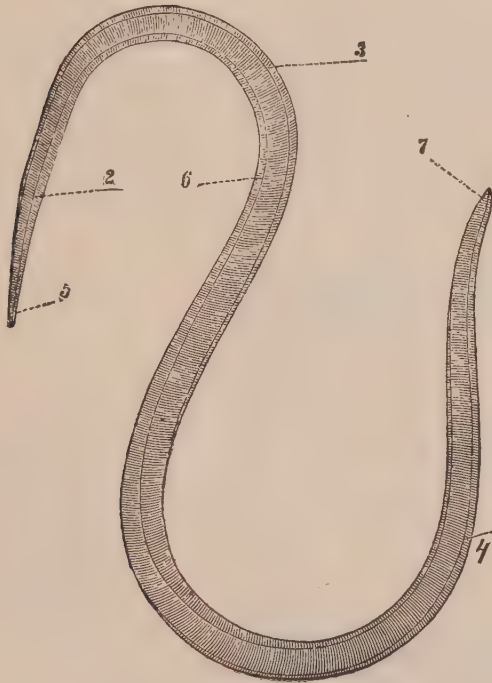


FIG. 34.

B

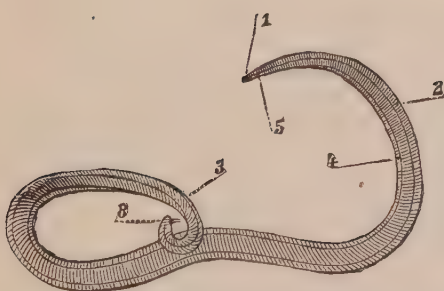


FIG. 35.

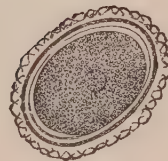


FIG. 34.—*Ascaris lumbricoides*. *a*, female; *b*, male, natural size. 1, cephalic end; 2, abdominal stripe; 3, left lateral stripe; 4, right lateral stripe; 5, porus excretorius; 6, female genital opening; 7, caudal end of the female with anal opening; 8, caudal end of the male with opening of cloaca and spicula.

FIG. 35.—Ovum of *Ascaris lumbricoides* with membrane and albuminous covering.

stools or are vomited, or if the ova can be detected in the fæces. If the ascaris is vomited, the patients often complain of very violent pain in the

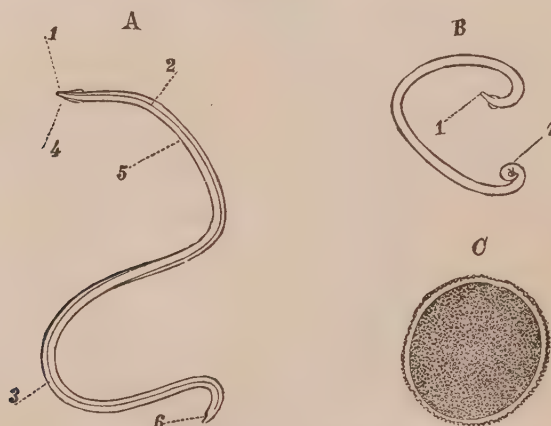
stomach. The number of worms vomited may be very considerable. Fauconneau-Dufresne reported a case in which a boy vomited 103 ascarides in the morning and 22 the same night.

Very dangerous conditions may be produced by the migration of the parasites. They sometimes enter the ductus choledochus, give rise to incurable and fatal jaundice, even pass into the hepatic ducts and produce hepatic abscess.

They sometimes form the nucleus of gall-stones. They have also been found in the ductus Wirsungianus. A number of cases have been reported in which the ascaris entered the larynx and produced suffocation. They may also pass into the bronchi and may give rise to gangrene and pulmonary abscess. Ascarides have been removed from the nose, the lachrymal canal, and the external auditory canal. As a matter of course, the latter is only possible if the drum membrane has been previously perforated.

If perforating ulcers are found in the intestines, ascarides are often

FIG. 36.



Ascaris mystax. A, female; B, male; C, ovum. 1, cephalic end; 2, abdominal stripe; 3, right lateral stripe; 4, porus excretorius; 5, female genital opening; 6, anus; 7, rolled-in caudal end with cloaca and spicula.

found in the peritoneal cavity on autopsy. In cases of intestinal fistula the worms are occasionally found in the uterus, vagina, or urethra, also in the pleural and pericardial cavities.

If a peritonitic abscess communicate with the intestines and also through the abdominal walls with the exterior, the round worms may pass externally through the fistula. They are sometimes found in the intestines in astonishing numbers. Fauconneau-Dufresne counted 5,126 which were passed by a boy of twelve within three months, although many evacuated by him were not counted.

IV. PROGNOSIS.—Complications produced by the migration of a parasite are so rare that the prognosis is not thereby rendered appreciably worse.

V. TREATMENT.—Ascarides are removed most effectually by the use of *santonine*:

R *Trochisci santonini*, āā gr. $\frac{3}{4}$.

D. t. d. No. x. S. Take one troche t. i. d.

It should be remembered, however, that certain individuals are apt

to suffer very readily from santonine poisoning (xanthopsia, yellow color of the skin, yellow urine, delirium, and even convulsions).

Among other remedies we may mention the following: *a.* Flores cinæ gr. 75, jalapæ gr. viiss. syr. simp. 3 viiss. M. D. S. To be taken in three portions. *b.* Natrium santonicum gr. viiss. *c.* Inf. flor. tanacetii 3 iij. : $\frac{2}{3}$ iiiss. *d.* Ol. tanacetii. *e.* Ol. terebinthinæ.

APPENDIX.

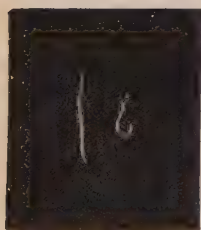
Ascaris mystax (round worm of the cat) has been found in man in a few cases. It is smaller than *ascaris lumbricoides*. The cephalic end presents a wing-like projection (vide Fig. 36).

Thread Worm. *Oxyuris vermicularis*.

I. ANATOMICAL CHANGES.—The length of the mature female is twelve mm., that of the male five mm.

The caudal end of the male is rolled in towards the abdominal surface,

FIG. 37.



Oxyuris vermicularis. On the left, the female; on the right, the male. Natural size.

FIG. 38.



Ova of *Oxyuris vermicularis*, obtained from the fæces. Enlarged 275 times.

and runs out into a fine end in both sexes, while at the anterior extremity the head is recognized as a very fine button.

The ova are 0.052 mm. in length and 0.024 mm. in width. They are oval, curved more on one side than on the other, contain granular contents with a distinct nucleus and nucleolus, and have a clear membrane. Their habitat is the entire large intestine. The rectum contains chiefly impregnated females in extremely large numbers.

II. ETIOLOGY.—If ova enter the stomach of the human being, the membrane is dissolved by the gastric juice, and gives exit to the embryo, which first remains in the small intestine and continues to develop. Here impregnation of the fully developed parasite takes place.

Hence, uncleanness may give rise to self-infection or infection of others. It is not true, however, that infection may take place by the migration of the oxyuris from the rectum to another individual sleeping in the same bed. Infection does not occur through the agency of drinking water because this rapidly destroys the ova.

Children and women are most frequently affected. The worm is also

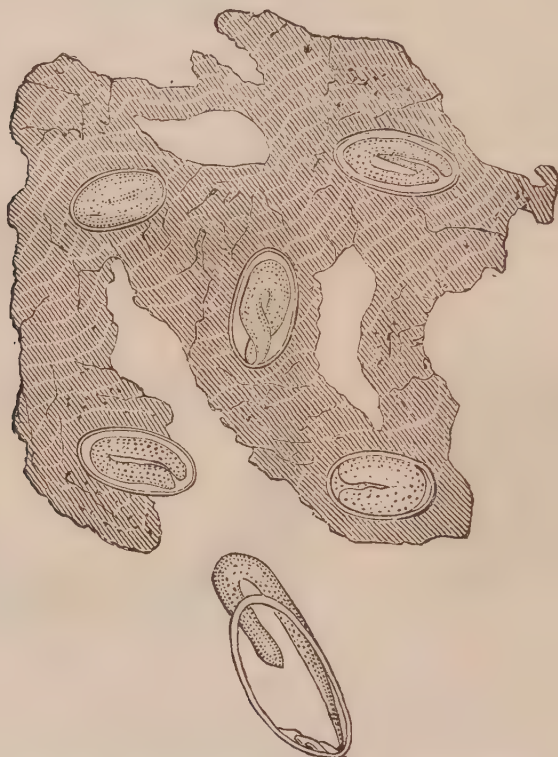
found often in filthy insane. Some persons suffer during their whole life, but the life of the individual thread-worm is very brief, so that in such cases self-infection must constantly be produced.

In a number of cases Zenker found the worms beneath the tips of the nails of human beings.

III. SYMPTOMS.—The local and general symptoms are similar to those produced by ascarides (vide page 152), but there are certain local peculiarities.

Violent itching at the anus is very often produced. Diarrhœa may

FIG. 39.



Integument infiltrated with ova of oxyuris. After Michelson.

occur occasionally. The parasites present a tendency to migrate; they leave the rectum and crawl across the perineum to the vagina, more rarely beneath the prepuce. In this manner they may produce leucorrhœa, balano-posthitis, and thus may give rise to onanism, even to nymphomania, spermatorrhœa, or prostatorrhœa.

Michelson described an eruption like eczema in the genito-crural fold, produced by inoculation with the ova of oxyuris (vide Fig. 39), but we must add that this was the result perhaps of an accidental infection with the ova.

IV. DIAGNOSIS.—The diagnosis is easy.

In the fæces we notice parasites in motion, often of a lively character. If they are not visible, the fæces should be examined microscopically for the ova.

V. PROGNOSIS.—The prognosis is good, but it seems as if certain individuals are especially apt to suffer infection.

VI. TREATMENT.—The object of treatment by the mouth is to remove the parasite from the small intestine. This is effected by the use of santonin lozenges, $\text{aa gr. } \frac{3}{4}$ three times a day, or the anthelmintics mentioned on page . . . Treatment per rectum consists of intestinal infusions to remove the parasites from the rectum. We may recommend an infusion of garlic, dilute vinegar, or soap and water. The infusion should be repeated daily for a number of days. The migration of parasites across the perineum may be prevented by smearing the vicinity of the anus with unguent. hydrargyri.

Whip Worm. Trichocephalus Dispar.

This is found in the cæcum, usually in small numbers. Its thick caudal extremity resembles the handle of a whip, while the thin cephalic extremity corresponds to the lash.

FIG. 40.



Trichocephalus dispar. The male above, the female below. Natural size.

The female is about fifty mm. in length, the male forty to forty-five mm. The caudal extremity in the male is rolled in towards the dorsal

FIG. 41.



Ova of *trichocephalus dispar.* Enlarged 275 times.

surface. The ova are oval in shape, 0.05–0.054 mm. in length, have a brownish color and a button-shaped projection at both poles.

The mode of infection is unknown. The parasite is found in almost

all countries. Copious discharge of the parasites has been observed in typhoid fever. Nothing is known regarding the symptoms to which they give rise.

Anchylostomum Duodenale.

(*Dochmius* s. *Strongylus duodenalis*.)

This is observed most frequently in the Orient and in the Tropics; it has also been found in northern Italy. Menche observed a case in the vicinity of Bonn, and a number of cases have also been reported in other localities.

The parasite is cylindrical in shape, the female ten to eighteen mm. long, the male six to ten mm. The cephalic end is curved towards the dorsal surface, and terminates with an oblique surface; the ova are oval, 0.05 mm. long, and 0.1023 mm. wide. They have granular contents and a clear membrane, but are often found in the fæces in a condition of beginning fission. With high powers the mouth is found to be armed with teeth. The parasite is probably ingested in the immature form in impure water. Its habitat is the small intestine. The buccal opening surrounds

FIG. 42.

FIG. 43.

FIG. 44.

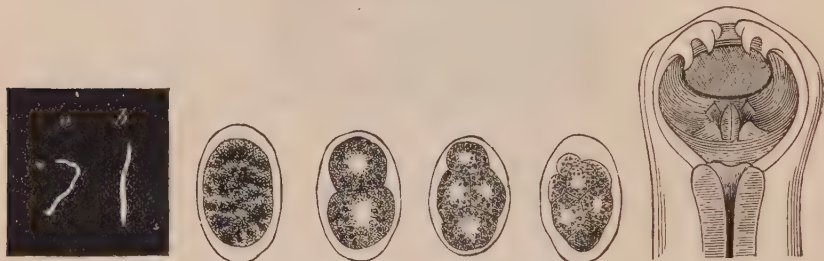


FIG. 42.—*Anchylostomum duodenale*. Male to the left, female to the right. Natural size.

FIG. 43.—Ova of *Anchylostomum duodenale* from human fæces. After Bugnion.

FIG. 44.—Buccal opening, armed with teeth, of *Anchylostomum duodenale*.

the intestinal villi, whence it draws blood. When the parasite seeks another part of the mucous membrane, the previous site continues to bleed. Hence the clinical symptoms of increasing anæmia. The passages from the bowels also contain blood. The entire symptom-complex has been termed anchylostomiasis. Griesinger first recognized the anchylostomum as the cause of tropical chlorosis. Perroncito attributes the anæmia of certain classes of workingmen (miners, brickburners, etc.), to the development of anchylostomum in the intestine. The diagnosis is possible if the parasite or ova are recognized in the stools. The prognosis is serious on account of the profound anæmia to which the disease gives rise. The treatment is the same in general as that of tapeworm, but it has been found that large doses of filix mas are the most effective. Treatment must sometimes be repeated a number of times.

Anguillula Intestinalis et Stercoralis.

Individuals living in the tropics sometimes suffer from dyspeptic conditions, diarrhœa, increasing emaciation, and anæmia. In 1876 Normand found anguillula in the stools of such patients. Perroncito also found them in the stools of the workingmen who suffered from the

Gotthard-tunnel anæmia. Seifert found the parasites in the vomited matters, so that they seem to be situated in the upper part of the intestine. This writer found that the best remedy is thymol, of which gr. xv.

FIG. 45.



Anguillula intestinalis. Enlarged 100 times. After Seifert.

was given every hour until twelve doses had been taken daily. The parasites have a lively movement in the fæces, and in Seifert's case were

FIG. 46.



Distomum crassum. Natural size.
After Leuckart.

FIG. 47.



Distomum heterophyes.

0.27 mm. long, and 0.016 mm. wide. The cephalic end was round, the caudal end pointed, the sexual opening was round.

APPENDIX.

Trichina spiralis will be discussed in Vol. IV. *Echinorhynchus gigas* was observed by Lambert in a child, by Welch in an Indian soldier, and by Lindmann in several individuals living on the Volga.

In a few cases, trematodes have also been observed in the intestinal tract of human beings, for example, *Distomum crassum* (Fig. 46) and *Distomum heterophyes* (Fig. 47). *Distomum hæmatobium* has also been observed, and may give rise to serious ulceration of the mucous membrane.

8. Intestinal Hemorrhage. Enterorrhagia.

I. ETIOLOGY.—Intestinal hemorrhage is the result of anomalies of the intestinal contents, of local diseases of the intestinal wall, or of general diseases. Obstinate constipation may give rise to hemorrhage if the excessively hardened fæces mechanically injure the mucous membrane.

The hemorrhage is usually insignificant, and appears in the shape of bloody dots and streaks, which are found upon the surface of the hard lumps of fæces. Ingested foreign bodies may act in a similar manner. Intestinal hemorrhage may also be the result of poisoning, and it should be remembered, that the immoderate use of cathartics acts in a similar manner. Finally, it may be produced by parasites (*Anchylostomum duodenale* and *Distomum hæmatobium*).

Among the local diseases the most important are the ulcerative changes of the intestinal mucous membrane. The comparative infrequency of hemorrhage in intestinal ulcers is owing to the fact that, if the ulceration progresses slowly, thrombi may form in the affected vessels. Injuries are sometimes the cause of intestinal hemorrhage. Rectal hemorrhages are not infrequently traumatic in character. Hemorrhage, though slight in extent, occurs not infrequently in enteritis.

It is relatively frequent in those forms which follow cutaneous burns. Bayer described a case in which it occurred in fatal erysipelas. Tumors of the intestinal mucous membrane are also causes of enterorrhagia. Furthermore, it is an important symptom in invagination, where it is the result of circulatory disturbances. Cases of a similar nature are observed in occlusion of the portal vein, and in hepatic, respiratory, and circulatory diseases.

Intestinal hemorrhages may also be the result of waxy degeneration of the vessels, embolism of the superior or inferior mesenteric arteries, or aneurisms of adjacent arteries which have ruptured into the lumen of the intestine.

Among general diseases, typhoid fever, dysentery and syphilis give rise to intestinal hemorrhage by ulceration of the mucous membrane. In intermittent fever, intermittent hemorrhages have been observed as the result of embolic occlusion of branches of the portal vein by melanine. Intestinal hemorrhages have been observed in typhus fever, though no ulcerations were present in the intestinal mucous membrane. Hemorrhage is rare in Asiatic cholera, frequent in yellow fever. It is observed occasionally in pyæmia and in septicæmia; also in the newborn as the result of puerperal infection. It is also observed in acute exanthemata of a hemorrhagic character, in hæmophilia, morbus maculosus Werlhofii, scurvy, purpura hæmorrhagica, and uræmia. Vicarious intestinal hemorrhage, *i. e.*, in the place of menstruation, has also been described.

Intestinal hemorrhage occurs most commonly in middle life. It is more frequent in males than in females.

II. ANATOMICAL CHANGES.—Bloody contents are found in the intestine, which is either distended with blackish-red clots or with tarry, foul-smelling masses. Sometimes the masses of fæces are hard and black, or the intestinal contents have the color of meat juice, and are partly mucous or muco-purulent.

In some cases the intestinal wall is extremely pale, in others the mucous membrane is suffused in places with blood or presents ulcerations. If the latter are the cause of hemorrhage, their blood-vessels contain thrombi, or water which is injected into the mesenteric artery will escape at the base of the ulcer. Ulcers, degenerating tumors, and foreign bodies may open directly into arterial or venous vessels, sometimes of considerable size.

In inflammations and circulatory stasis, the chief factor is the increased blood pressure, which cannot be resisted by the perhaps very

slightly changed wall of the blood-vessel. In conditions of blood dissolution we must assume changes in the walls of the vessels, which become unusually permeable. The hemorrhage may be arterial, venous, or capillary; even the latter may be very profuse and indeed fatal.

The other organs are usually very pale. If extensive previous hemorrhages have occurred, we not infrequently find fatty degeneration of the heart, liver, kidneys, pancreas, and glandular cells of the stomach and intestines.

III. SYMPTOMS.—Only those symptoms will be considered which are independent of the primary disease.

Intestinal hemorrhage sometimes gives rise to no other signs than those of internal hemorrhage. The patients grow exceedingly pale, the features are peaked, the skin cool, the pulse accelerated or scarcely perceptible, the heart sounds feeble; they complain of spots before the eyes and ringing in the ears, nausea, vomiting, dizziness, and fainting spells. Finally, death may occur, although not a drop of blood has escaped externally. An abnormal area of dulness, corresponding to the accumulation of blood, is occasionally found in the abdomen.

The chief manifest symptom is the evacuation of bloody stools. Sometimes these are purely bloody, forming loose blackish clots, more rarely fluid and bright red. In other cases the blood and fæces are intimately mixed, and the evacuations form blackish, tarry masses, often emitting an intolerable stench. The bloody masses are sometimes hard and intensely black. In hemorrhages from the rectum the blood appears on the surface of the fæces, while the inside of the mass of fæces is free from blood. In dysentery, the stools have the color of meat juice and contain muco-purulent or purulent masses.

The evacuations are sometimes attended with tenesmus. I have also observed obstinate constipation associated with fever; the administration of cathartics led to the evacuation of astonishingly large masses of bloody fæces, and the tenesmus and fever soon ceased.

On microscopical examination of the stools, the red blood-globules are sometimes found unchanged, sometimes swollen, sometimes discolored and in a condition of degeneration.

Nothnagel showed that microscopical examinations of the fæces sometimes reveals premonitory signs. In cases of typhoid fever, he found small amounts of blood in the fæces twelve to thirty-six hours before an extensive internal hemorrhage.

Hematemesis may be expected in those cases alone in which the hemorrhage takes place in the duodenum.

Physical examination of the abdomen must be performed very cautiously, in order to avoid an exacerbation of the hemorrhage. We should pay attention to dulness, increase in the area of dulness, and increased feeling of resistance.

Some patients state that they experience the sensation as if a warm fluid were flowing into the abdomen.

If the hemorrhage cannot be checked, or if it is too extensive, death will ensue. Under other circumstances oedema may develop and sometimes even mild albuminuria. In one case Traube observed death from oedema of the glottis, but this was complicated by laryngeal ulcerations.

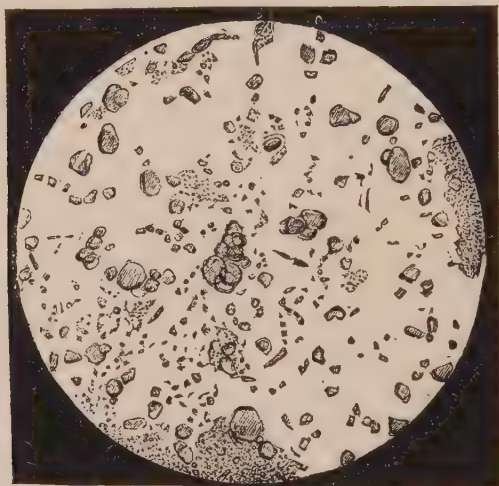
IV. DIAGNOSIS.—The diagnosis involves an answer to the following four questions: *a.* Is blood present in the stools? *b.* Is the blood de-

rived from the intestine? *c.* Does it come from the small or large intestine? *d.* What are the special causes of the hemorrhage?

a. In very obstinate constipation the fæces may assume a brownish-black color; if there is a copious admixture with bile the stools may also have a blackish-green color, which is mistaken for blood. A similar mistake may be made after the use of iron preparations and bismuth, or the passage of undigested red fruit. In doubtful cases the microscope may be resorted to; finally, we may resort to spectrum analysis and the formation of Teichmann's blood crystals.

b. Bloody stools indicate intestinal hemorrhage only when hemorrhages from the nose, pharynx, œsophagus, or stomach may be excluded. Bloody stools are also observed in the new-born after the operation for hare-lip, or the ingestion of blood during delivery or as the result of lesion of the nipples of the mother.

FIG. 48.



Changed red blood-globules from enterorrhagia, five days after the beginning of the hemorrhage. Enlarged 275 times.

c. Inspection and palpation occasionally suffice to decide the location of the hemorrhage (lower part of the rectum). The causes should also be taken into consideration. For example, intestinal hemorrhages after burns always originate in the duodenum; dysenteric hemorrhages in the large intestine, etc. If the blood is situated only upon the surface of the fæces, the hemorrhage must be referred to the large intestines. In some cases, the occurrence of abdominal dulness is useful in diagnosis.

d. The etiology depends upon the clinical history and associated symptoms. In latent intestinal hemorrhage, the diagnosis depends upon the symptoms of internal hemorrhage and the exclusion of a lesion of other organs.

V. PROGNOSIS.—The prognosis depends upon the causes and the profuseness of the hemorrhage. In many cases, the prognosis is unfavorable on account of the primary disease. Occasionally a favorable influ-

ence is attributed to the hemorrhage; this is especially true of typhoid fever.

If the hemorrhage exceeds a certain amount, however, it cannot possibly be regarded as a favorable event.

VI. TREATMENT.—Prophylactic measures must be taken into consideration in ulceration of the intestines. The diet and stools must be carefully regulated in order to avoid irritation of the surface of the ulcers. When the hemorrhage occurs, the treatment should be the same in general as in hemorrhage from the stomach (vide page 55).

The patient should be kept absolutely quiet, and receive only fluid, cool food, for example, milk and ice, red wine and ice, etc. An ice-bag should be applied to the abdominal walls, a subcutaneous injection of ergotinum Bombelon (one-half syringe-ful mixed with an equal amount of water) should be made, and liquor ferri sesquichlorati given internally. If the intestinal movements are vigorous, we should give several large doses of opium in rapid succession. In rectal hemorrhage, we may inject ice-water, which may be mixed with astringents, for example, tannic acid (gr. xxx.); nitrate of silver (gr. viiss.); liquor ferri sesquichlorati (10 drops). In collapse, we should give strong wine, brandy, musk (gr. ivss. every hour), or subcutaneous injections of camphor (gr. xv., Ol. Amygdal., 3 iij., one syringe-ful three times a day).

APPENDIX.

Melæna Neonatorum.

(Apoplexia Neonatorum Intestinalis.)

I. ETIOLOGY.—The term melæna is now applied only to hæmatemesis and enterorrhagia in the new-born. In melæna vera the blood is derived from the stomach or intestine; in melæna spuria the blood has been ingested. We will now discuss only melæna vera. In a few cases gastric ulcers have been observed in the stomach and duodenum. Hænoch described one case in which the ulcer was situated in the œsophagus immediately above the stomach. In some cases, we find merely congestions of the intestinal mucous membrane. In another group, there are signs of general dissolution of the blood. Opinions differ in regard to the ulcerative forms of melæna, whether we have to deal with the results of embolism or of hemorrhagic infiltration of the mucous membrane. According to Bohn, the ulcers are the result of occlusion and suppuration of the gastro-duodenal glands. Steiner claims to have noticed fatty degeneration of the vessels of the mucous membrane. In many cases, no cause whatever could be discovered, the disease attacking well-developed children of healthy parents. In some cases, difficult labor had been regarded as the cause of melæna, and it is said to be observed with special frequency in asphyxiated children. Finally Kiwisch and Landau attach great importance to the too early ligation of the funis. The disease is not frequent. Hecker observed it once amongst five hundred births, Genrich only once in one thousand births.

II. ANATOMICAL CHANGES.—The anatomical changes, in so far as they affect the wall of the intestine, have been previously mentioned. The lymph follicles of the intestinal mucous membrane have been found swollen, and even follicular ulcers have been described. Enlargement of the spleen has been observed a number of times, and likewise changes in the liver.

III. SYMPTOMS.—The symptoms sometimes develop suddenly, or are preceded by prodromata (pallor of the skin, low temperature, disappearance of the pulse, increasing apathy), which must be attributed to an internal hemorrhage. The first manifest symptoms often appear a few hours after birth, but generally on the second day. In rare cases, they have been observed in the second week, and even the beginning of the third week. As a rule, the first manifest symptom is the passage of bloody stools, and then hæmatemesis soon occurs. Silbermann collected forty-two cases, with the following results:

Hemorrhage from the stomach and intestines, .	25 times.
“ “ “ intestines alone, .	10 “
“ “ “ stomach alone, .	7 “

The blood is sometimes dark and sometimes bright-red. Occasionally only one or a few bloody stools are passed; in other cases, the hemorrhages are repeated, and may even continue for several days. Symptoms of increasing anæmia develop and prove fatal. Even in favorable cases, dangerous weakness may persist for months, and in some cases a tendency to gastric and intestinal diseases continues for the remainder of life.

IV. DIAGNOSIS.—The symptoms of melæna neonatorum are so sharply defined that it is scarcely possible to mistake it for other conditions.

V. PROGNOSIS.—The prognosis is always grave, and, on the average, fifty per cent of the cases prove fatal. If the hemorrhage has lasted longer than thirty-six hours, recovery is exceptional.

VI. TREATMENT.—The funis should not be tied until pulsation is no longer felt and respiration is regularly performed. During an epidemic of puerperal fever, the infant should be removed from the mother in order to avoid as much as possible the danger of infection.

After melæna has developed, we may apply compresses to the abdomen, give Ergotinum Bombelon (one-quarter syringeful) subcutaneously, and internally give a few drops of wine.

1. *Hæmorrhoids, Phlebectasia hæmorrhoidalis.*

(*Hæmorrhoidal disease. Piles.*)

I. ETIOLOGY.—Hæmorrhoids are a dilatation of the hæmorrhoidal veins, and sometimes occur in a diffuse manner, sometimes in the shape of individual varices. The causes may be purely local and confined to the rectum. For example, in individuals who suffer from obstinate constipation, it is generally assumed that the retained fæces impede the circulation in the lower part of the rectum, but Duret attaches chief importance to excessive straining during defecation. Hæmorrhoids are sometimes the result of protracted proctitis, inasmuch as the submucous tissue becomes more yielding, and the veins contained in it undergo dilatation. Not infrequently hæmorrhoids and proctitis are the result of the same cause, and, in addition, hæmorrhoids may give rise to secondary proctitis. Cancer and stricture of the rectum are often followed by the development of hæmorrhoids. They are occasionally the result of affections of the uterus, ovaries, or prostate, when these organs press upon surrounding parts and interfere with the circulation. The obstruction to the circulation is sometimes situated higher, as in stasis of the portal circulation, whether the result of portal thrombosis, compression of the

portal vein, or diseases of the liver itself. Affections of the circulatory and respiratory apparatus act in the same manner as soon as they give rise to stasis in the inferior vena cava.

Hæmorrhoids are often found in plethoric individuals, who are fond of the pleasures of the table and averse to bodily exercise. In these cases circulatory disturbances in the portal vein are usually regarded as the cause of the disease. As a rule, hæmorrhoids develop between the thirtieth and fiftieth years of life. They are very exceptional in childhood, but Lannelongue reported a case in which they developed a few days after birth. They are more frequent in men than in women. Their development is favored by sedentary occupations; excesses in venery, constant sitting on benches, abuse of alcohol, constant horse-back riding, are also predisposing factors, partly because they produce congestion of the pelvic organs and partly because they favor stasis in the rectal veins. I have often observed the disease in old musicians who play on wind instruments.

The facility with which the rectal veins yield to obstructions in the circulation is owing particularly to the fact that they are destitute of valves. The portal vein also is destitute of valves, so that all disturbances of the portal circulation are very readily propagated to the hæmorrhoidal veins. In addition, we must take into consideration the action of the force of gravity.

II. ANATOMICAL CHANGES.—We distinguish between external and internal hæmorrhoids. The former are situated outside of the sphincter ani, while the latter are situated above this muscle, and are only recognized by digital examination or by means of the speculum. In some cases, a portion of the varix is situated outside of the anus, the other part within.

There may be a diffuse dilatation of the rectal veins, so that in the case of external hæmorrhoids the rectum is surrounded by a bluish prominent ring; in other cases we find circumscribed dilatations, which vary considerably in number and size (from that of a lentil to that of a pigeon's egg, and even an apple). They may be round, flattened, or irregularly angular. Internal and external piles may be associated with one another. As a rule, internal hæmorrhoids are also situated near the sphincter ani, but Petit described a case in which they extended to the sigmoid flexure.

Secondary changes may develop in the hæmorrhoids themselves or in their vicinity. A relatively favorable process is the formation of thrombi, which become organized, and thus produce spontaneous occlusion of the dilated portion of the veins. This may also result in calcification and the formation of venous calculi (phleboliths). If the varices are extensive and closely aggregated, the septum between the adjacent ones sometimes disappears, so that several large spaces partly coalesce, and thus constitute a sort of cavernous tumor. A frequent complication is thickening of the submucosa of the rectum and chronic proctitis, which is generally manifested by a profuse puriform secretion. Not infrequently there is inflammatory hyperplasia of the periproctal connective tissue. Sometimes an acute inflammation develops, and results in the formation of an abscess. This may rupture externally into the rectum, or in both directions, thus giving rise to external, internal, or complete rectal fistula.

In external hæmorrhoids the covering of skin is sometimes as thin as paper, sometimes abnormally thickened. Internal hæmorrhoids are oc-

casionaly pedunculated, particularly if they have been extruded from the anus.

III. SYMPTOMS.—Dilatation of the hæmorrhoidal veins not infrequently exists without giving rise to symptoms; the latter are not produced until the piles have reached a certain size, and thus give rise to mechanical disturbances. On the other hand, patients sometimes complain that they have suffered for years from hæmorrhoids, although nothing is discovered on the most careful examination.

Many patients complain of an annoying sensation of itching, burning pain, or increased heat at the anus. These symptoms increase after a heavy meal, constant sitting or riding, and excesses in *Baccho et Venere*. Not infrequently the patients are annoyed by the sensation of a foreign body in the region of the anus.

The symptoms become more severe when the piles narrow the exit from the rectum, and thus interfere with the stools. The patients are often tortured by the most violent pains during defecation, and in sensitive individuals this may give rise to syncope and convulsions. Many patients delay the passages as long as possible. This may result in distention of the abdomen, singultus, nausea, vomiting, difficulty of breathing, palpitation of the heart, rush of blood to the head, etc.

Hæmorrhoids are sometimes manifested by nothing beyond chronic blennorrhœa of the rectal mucous membrane. The patients must often go to stool, and pass muco-purulent or almost purely purulent masses, which may or may not be mixed with fæces. Dots and streaks of blood are occasionally noticeable, and the disease may be mistaken for dysentery.

Hemorrhages are very often observed in these patients. The are not always the result of rupture of the varix, but in the majority of cases are of capillary origin. Hemorrhage very rarely occurs without the previous existence of other symptoms. It is usually preceded by the so-called *molimina hæmorrhoidalis*, which consist of a feeling of increased tension and pain in the anus, a rush of blood to the head, palpitation of the heart, etc. The amount of blood discharged may vary from a few tablespoonfuls to several pounds. Sometimes a single hemorrhage occurs, sometimes the hemorrhages are repeated for days.

In many patients this is followed by a feeling of great relief. As the blood is derived from the lower part of the rectum it is usually but little changed. It merely coats the fæces, and not infrequently is discharged in an unmixed condition. The loss of blood is rarely so considerable as to threaten life, but if the hemorrhages last for a long time, or are repeated in rapid succession, they may give rise to serious conditions of anæmia. The patients are pale, short of breath, feeble, suffer from œdema, and occasionally from slight albuminuria. Dilatation of the right ventricle sometimes occurs, and we may find anæmic cardiac murmurs, the *bruit de diable* in the jugular veins, and the arterial sounds in smaller peripheral arteries. Very painful conditions are produced by prolapse and incarceration of internal hæmorrhoids. After violent straining, internal piles may be forced through the sphincter to the outside. If the sphincter ani now undergoes spasmodic contraction, the prolapsed hæmorrhoids are prevented from returning. Such conditions cause extremely violent pain. The patients groan aloud, the brow is covered with perspiration, the pulse becomes small and frequent, and in some cases syncope and convulsions make their appearance. If the hæmorrhoids cannot be replaced, inflammation and

gangrene set in, and this may be followed by serious conditions, for example, pyæmia.

The duration of hæmorrhoids is usually short if their causes are transitory in character, as for example, in pregnancy. In the majority of cases, however, the disease is chronic, and usually lasts for life. Pyschical depression often develops, but is the result of the associated gastric and intestinal affection rather than of the hæmorrhoids.

IV. DIAGNOSIS.—External hæmorrhoids are readily recognized on inspection, internal ones by digital examination and the use of the speculum. The following mistakes in diagnosis may be made: *a.* With folds of skin at the anus (the bluish appearance, tension, and spherical shape of hæmorrhoids are wanting); *b.* With condylomata (other signs of syphilis are usually present upon the genitals, or mucous membranes); *c.* Rectal cancer (cachexia present in these cases).

V. PROGNOSIS.—Prognosis as regards life is good, but, as a rule, we cannot hope for the permanent disappearance of the disease.

VI. TREATMENT.—Prophylactic measures must not be omitted. The remedies recommended on page 122 should be given to individuals who suffer from constipation. Gourmands should be restricted in diet, should eat less meat and more vegetables, and should avoid rye bread, strong wine, coffee, or tea. Stasis should be prevented as long as possible in diseases of the respiratory organs, heart, liver, or portal vein.

After hæmorrhoids have developed, care should be taken to secure an easy and soft evacuation from the bowels. The following prescriptions enjoy special repute:

℞ Pulv. liquirit. comp.,	• • • • •	3 i.
D. S. One or two teaspoonfuls to be taken at night.		
℞ Sulphur. depurat.,		
Kali bitartrat.,	• • • • •	āā 3 vi.
Rhizomat. zingiber.,		
Rhizomat. calami,	• • • • •	āā 3 i.
M. D. S. One teaspoonful to be taken at night.		

As a matter of course, the dietetic measures previously referred to must also be adopted. Good results are obtained in many cases from the use of the waters of Carlsbad, Kissingen, Homburg, Marienbad, Rohitsch, Tarasp, etc. In very plethoric patients excellent results are often secured by the sulphur waters of Weilbach, Neuendorf, Eilsen, Baden near Vienna, Schinznach. In anæmic individuals we may employ mild acidulous iron springs, for example, Elster, Franzenbad, Cudowa, or the alkaline-muriatic acidulous waters, for example, Ems, Soden, Cannstatt, Baden-Baden, etc. Whey cures and grape cures are indicated in cases of obstinate constipation and marked mental depression.

Specially prominent symptoms not infrequently require treatment. In order to prevent irritation of large external piles, they may be anointed with o. amygdalar., ol. cacao, etc. If there is violent pain in the region of the anus, we may order suppositories of opium, morphine, belladonna (vide page 112). Relief is also afforded, as a rule, by compresses of ice-water, lead wash, or liq. alumin. acetic. (one per cent). In rectal blennorrhœa, intestinal infusions should be made with cold water and astringents, for example, nitrate of silver (gr. iss.—viiss. at each infusion), tannicacid (gr. iiiss.—xv.), etc. Hemorrhages require treatment only when they are unusually severe. We may inject ice-water, pure or mixed with astringents, or the rectum may be tamponed with cotton and Ergotinum Bombelon injected subcutaneously (one-half syringeful). Lan-

dowski recently extols repeated hot sitz-baths (to 40°) and rectal injections of hot water. If the hæmorrhoids are prolapsed, the patient should be placed in the knee-elbow or lateral position, and an attempt made to replace the piles with the oiled finger, or a piece of linen dipped in oil. After reposition of the piles, renewed prolapse should be prevented by a light bandage to the anus. If this is associated with severe pain, the bandage may be sprinkled with laudanum, or smeared with an ointment of morphine or belladonna. If the piles cannot be replaced, they may be punctured or incised, and the attempt at reposition then renewed. If gangrene sets in, we may apply moist, warm poultices to facilitate the removal of the necrotic portions. In cases of violent molimina hæmorrhoidalis, five to ten leeches may be applied to the anus, and the hæmorrhage kept up by allowing the patient to use a vessel in which hot water has been placed.

Radical relief can only be effected by surgical operation, but for the discussion of the subject we must refer the reader to text-books on surgery.

10. *Nervous Intestinal Pain. Enteralgia (Colic. Enterodynia. Neuralgia mesenterica).*

I. ETIOLOGY.—The term colic is applied only to those cases of intestinal pains which are independent of anatomical changes in the intestinal walls. The disease is a neurosis which sometimes occurs from peculiar changes in the intestinal contents, sometimes as an independent neurosis.

The following changes in the intestinal contents must be taken into consideration: *a. Coprostasis.*—The process is here chiefly mechanical, inasmuch as the hard masses of fæces irritate the mucous membrane; in addition, the intestine is markedly distended. A special form is colica meconialis of the new-born, which occurs when the meconium is retained for some time after birth. *b. Foreign bodies* in the intestines sometimes give rise to colic (a coil of ascarides, tape-worm, ingested sharp bodies, faecal calculi, and gall-stones, which pass through the intestines). *c. Decomposing food*, for example, sour beer, unripe fruit, fermented milk. *d.* In some cases, certain physical characteristics of the food are injurious; in others, there are peculiar idiosyncrasies. Thus a cold drink may be the cause of colic, and some individuals suffer whenever they partake of fish, oysters, certain vegetables, or fruits, etc. *e.* A frequent form is *wind colic* (flatulent colic), which is the result of the excessive development of gas and distention of the intestinal walls. It is found particularly in children. *f. Toxic colic* is observed after the use of certain cathartics, for example, senna, castor oil, etc. This is also noticed in lead and copper poisoning.

Colic is observed as a neurosis, in the stricter sense, in hypochondria and hysteria, and also in organic diseases of the nervous system.

It develops not infrequently as a reflex symptom in disease of the uterus, ovaries, liver, or kidneys. It has been observed immediately before an attack of gout, or instead of the seizure. In many cases, it is the result of a cold.

II. SYMPTOMS.—The chief symptom of colic is intestinal pain. This is referred usually to the region of the umbilicus, but often radiates into the back, chest, testicles, and thighs. It often remains in one locality. In other cases, it moves from place to place, and is associated with borborygmus, or with perceptible protrusion of individual parts of the intestines. The pain gradually assumes greater and greater intensity, and then

gradually subsides. More rarely the pains are lightning-like in character. They are described as cutting, twisting, sticking, or as if the intestine were violently stretched. The patients groan aloud, the face grows pale, the skin becomes cool and often covered with perspiration, the pulse is usually slow and hard. Sometimes the pains last a few seconds, and at other times many minutes. Many patients assume peculiar positions of the body. The thighs are drawn up on the abdomen, or the hands are pressed against this region, or the abdomen is pressed against a firm object, or the patient assumes abdominal decubitus.

The abdomen is sometimes hard or sunken, sometimes it is tympanitic.

In certain cases, peristaltic movements of distended loops of the intestines can be seen beneath the abdominal walls. Pressure often, though not constantly, relieves the pain; indeed, in some patients the sensitiveness of the abdominal walls is so great that a suspicion of peritonitis may be aroused.

Reflex symptoms are often noticed in other organs, for example, singultus, vomiting, asthmatic symptoms, palpitation of the heart, strangury, tenesmus, etc. The testicles are not infrequently drawn strongly upwards, and the levator ani is spasmodically contracted. Priapism and pollutions are observed in rare cases, cramps in the calves, fainting-spells, and general convulsions develop in others.

In many cases, the colic ceases suddenly after vomiting, eructations, or the discharge of flatus or fæces. Death occurs only in exceptional instances. Oppolzer observed it as the result of rupture of the intestine from excessive distention by the gas, and Wertheimer noticed death in convulsions in another case.

III. DIAGNOSIS.—The disease may be mistaken for: *a.* Rheumatism of the abdominal muscles. In this disease, the pain often changes its locality, lasts for a longer time, and presents no distinct exacerbations and remissions. The pain is situated nearer the surface, and is produced on slight contact with the abdominal walls. *b.* Lumbo-abdominal neuralgia. Valleix's pressure points are present in this disease. *c.* Nervous pain in the integument of the abdominal muscles in hysterical patients. According to Briquet, this is rapidly relieved by the faradic current. *d.* Circumscribed peritonitis. Dulness on percussion and fever are present in this affection.

IV. PROGNOSIS.—The prognosis is almost always good, but relapses occur very frequently.

V. TREATMENT.—The colic itself may be treated by the subcutaneous injection of morphine, or the internal administration of chloral hydrate or opium (gr. ss., three powders at intervals of half an hour). Constipation does not contraindicate the administration of opium. The abdomen should be covered with a warm poultice (flaxseed), and one or more cups of warm carminative tea given.

Among numerous other remedies which have been recommended we may mention belladonna and its preparations. Good results have also been obtained from the use of electricity: a vigorous faradic current, one pole in the rectum, the other labile upon the abdominal walls for five to ten minutes.

APPENDIX.

1. Trousseau called attention to disturbances in the motor activity of

the intestinal nerves. Nervous constipation or diarrhœa may be produced according as the activity is diminished or increased.

In some individuals fright or bashfulness exerts a laxative effect. In others certain articles of diet give rise almost immediately to diarrhœa, so that this can hardly be explained except as the result of increased peristalsis from purely nervous causes. Such cases are observed particularly in neurasthenic, hysterical, and hypochondriacal individuals, but occasionally as reflex symptoms of diseases of the female sexual organs. The intestinal affection can only be relieved by treatment of the primary disease.

2. A few words with regard to embolism of the mesenteric artery. Scattered emboli in the smaller branches often produce no serious results, because the numerous collateral vessels compensate the obstruction to circulation. But if the main trunk or several adjacent branches are occluded, that portion of the intestine whose circulation is interfered with undergoes hemorrhagic infarction, and even becomes necrotic. Extravasation also occurs between the layers of the mesentery. Peritonitic symptoms are not infrequent.

The emboli are derived generally from vegetations of the valves of the left side of the heart, more rarely from aortic aneurisms.

The patients are suddenly seized with violent colicky pains, they grow pale and collapse occurs. The signs of peritonitis develop, occasionally the extravasation in the mesentery is felt as a tumor. In addition, blood is evacuated from the bowels. The prognosis is grave, although two cases of recovery have been reported. Treatment: Stimulants, morphine in violent pain, and intestinal infusion of ice-water against the hemorrhage, if this appears in the stools as fresh blood, and therefore indicates embolism of the inferior mesenteric artery; otherwise Ergotinum Bombelon should be given subcutaneously.

SECTION V.

DISEASES OF THE LIVER.

a. Diseases of the Biliary Passages.

1. Stenosis and Occlusion of the Bileducts. (Obstructive Jaundice. Absorption Jaundice. Icterus Hepaticus s. Mechanicus).

I. ETIOLOGY.—In stenosis or occlusion of the bile-ducts the bile must accumulate above the obstruction. As it is secreted under very slight pressure, insignificant disturbances in the bile-ducts will produce stasis. The bile is then absorbed in greater part by the lymphatic vessels of the liver, to a less extent by the blood-vessels, and is thus carried into the general circulation. The biliary coloring matter in the blood then gives rise to jaundice (icterus). As a rule, occlusion of the bile-ducts can be recognized only by the existing jaundice.

The causes of the stenoses of the bile-ducts may be situated within or without the liver.

In the first event they affect chiefly the finer ducts within the hepatic parenchyma, not infrequently the intralobular biliary capillaries, while extra-hepatic causes affect the large excretory ducts (ductus hepaticus, choledochus, cysticus).

Absorption jaundice is a frequent symptom of many hepatic diseases. It is absent in uncomplicated fatty and waxy liver. In one and the same disease icterus is sometimes absent, sometimes very marked, according to the degree of implication of the biliary passages. If only small amounts of bile are absorbed it will be excreted by the kidneys with sufficient rapidity to prevent its accumulation in the blood.

Absorption jaundice sometimes depends indirectly upon diseases of the liver, for example, in hepatic cancer when the periportal glands are enlarged and compress the excretory ducts.

Diseases of the biliary passages themselves not infrequently give rise to stenosis and occlusion, and thus to jaundice. One of the most frequent causes is catarrh of the bile-ducts, in which a profuse accumulation of secretion constitutes the obstruction.

Ebstein showed that the catarrhal affection may be confined under certain circumstances to the intra-hepatic biliary passages, so that the origin of the jaundice can only be determined by microscopical examination of the finer ducts. Virchow showed that not infrequently marked inflammatory swelling of the walls of the bile-ducts may disappear after death. Croupous inflammation of the bile-ducts rarely gives rise to absorption jaundice because the former disease is in itself infrequent.

The bile-ducts are often obstructed by foreign bodies, usually gall-stones, in other cases parasites. For example, echinococcus vesicles may rupture into the biliary passages, or multilocular echinococci may take their origin in this locality. Distomum of the bile-ducts may also give rise to stenosis. Occlusion of the ductus choledochus may also be produced by ascarides, cherry-stones, etc. Whether occlusion may be produced by inspissated bile, has not been positively decided.

Cicatricial strictures (usually the result of ulceration by gall-stones) occasionally produce stenosis, sometimes even obliteration of the bile-ducts. Similar conditions have been observed after catarrh of the biliary passages attended by epithelial desquamation, and after infectious diseases.

In a few cases, cancer, lipoma, or polypi of the walls of the bile-ducts are the cause of the obstruction to the flow of bile. According to Schueppel, chronic inflammation of the excretory ducts and surrounding connective tissue may also produce stenosis.

Congenital obliteration of the bile-ducts from foetal inflammation has been observed in several cases.

In not a few cases the stenosis is the result of an affection of adjacent organs. Thus, catarrh of the duodenum very often gives rise to occlusion at the point of entrance of the ductus choledochus, either by a plug of catarrhal secretion and desquamated epithelium, or by catarrhal swelling of the mucous membrane itself. Cancer and cicatrizing ulcer in the duodenum may act in a similar manner. The bile-ducts sometimes undergo compression in consequence of cancer of the stomach, and similar changes may be produced by distention of the colon with fæces, cancer of the colon, the pregnant uterus, large tumors of the uterus or ovaries, of the pancreas, omentum, mesentery, retroperitoneal glands, aneurism of the hepatic or mesenteric arteries, renal tumors, and floating kidney. In some cases the obstruction is the result of chronic peritonitis which has given rise to the formation of retracting callosities.

Obstructive jaundice has also been observed in right diaphragmatic pleurisy and acute peri-hepatitis. As is well known, the movements of the diaphragm exercise a very material influence upon the flow of bile, inasmuch as the inspiratory movements increase the pressure to which the liver is subjected, and thus facilitate the flow of bile into the intestine. But since the movements of the diaphragm are diminished or abolished in the diseases mentioned, the bile may accumulate in the intra-hepatic ducts, and thus pass into the general circulation.

II. SYMPTOMS.—If sufficient amounts of the biliary coloring matter enter the blood, the skin will assume a yellow color; this may vary from bright sulphur or orange-yellow to saffron-yellow, greenish-yellow, bronze and even blackish-yellow. Apart from the intensity of the stasis of bile, the color of the skin, as a rule, is so much darker, the older the patient, the thicker the epidermis, and the longer the jaundice has lasted.

The mucous membrane also assumes a yellow color. The white color of the sclera disappears, and gives place to yellow. On the mucous membrane of the lips, tongue, and buccal cavity, pressure with the fingers must first be exercised in order to squeeze out the blood before the icteric color makes its appearance. If the mouth is widely opened, two icteric stripes will be seen on the soft palate and the posterior part of the hard palate, because the tension produced by opening the mouth has caused anæmia of these localities.

If sudden, complete occlusion of the ductus choledochus or hepaticus has occurred, three days generally elapse before the first icteric changes become visible in the integument. The first indication generally appears in the sclerotic conjunctiva, although in some cases this remains unchanged despite marked jaundice of the skin. The yellow color then appears in those parts of the skin which possess a thin epidermis and abundant vessels (naso-labial folds and lips, forehead, chest, abdomen, and back). The yellow color at first depends upon the presence of the

biliary coloring matter in the plasma of the blood, but later, this is deposited in the deeper layers of the rete Malpighi.

Very annoying itching of the skin (*pruritus cutaneus*) is observed in not a few cases. It generally appears after the jaundice has lasted for some time, more rarely at the beginning of the disease (Graves reported a case in which it even preceded the jaundice). This symptom may sometimes almost drive the patient to distraction. It occasionally occurs only at night, and indeed, as a rule, it is intermittent. It usually occurs only in circumscribed spots, particularly in the palms of the hands and soles of the feet, or between the fingers and toes. Scratch marks are generally present upon the skin. The *pruritus* sometimes disappears unexpectedly, although the jaundice continues unchanged. It has been attributed to irritation of the cutaneous nerves by the biliary pigment which has accumulated in the epidermis. A well-known physicist who suffered from intense jaundice assured me that, shortly before the beginning of the itching, he experienced in the affected parts a sensation of cold; on a number of occasions this was also recognized objectively. This will suggest the idea that the symptom is the result of vaso-motor disturbances. Eruptions may make their appearance upon the skin, sometimes of an erythematous character, sometimes similar to urticaria.

English authors call attention to the occasional development of *xanthelasma* which is confined not alone to the skin, but may also extend to the mucous membrane of the buccal cavity. The perspiration very often presents an icteric color. Andral even reports that he observed icteric perspiration in a patient, although the integument and conjunctivæ appeared to be unaffected.

According to some authors urinary changes precede the cutaneous symptoms, but in our experience this is exceptional. The kidneys serve to remove from the body the biliary coloring matter which has accumulated in the blood. Its presence in the urine causes the latter to assume an unusually dark, reddish-brown appearance resembling that of porter. It is distinctly dichroitic, *i. e.*, it glitters in reflected light, especially at the sides; after standing for some time it not infrequently assumes a greenish color throughout. When shaken, a yellow froth makes its appearance, and remains for an unusually long time.

Traces of albumin are sometimes present and casts are constantly found in the urine. The latter are hyaline, not infrequently covered with fat granules, and occasionally bile-stained epithelium cells from the urinary tubes.

The presence of bile pigment in the urine may be shown by Maréchal's or Gmelin's test. *a.* In carrying out the former, the urine is poured into a tube, and a few drops of tincture of iodine are added. If the urine is shaken, it assumes a beautiful emerald green color when bile pigment is present. *b.* Gmelin's test is employed in the following manner: Commercial nitric acid is poured into a test-tube, and the urine is then allowed to flow slowly upon its surface. If the urine contains bile pigment a series of colored rings will appear at the layer of contact, the uppermost green, then blue, then violet-red, then orange, and finally yellow. The green color alone is conclusive, since a brown ring is obtained in many concentrated urines. O. Rosenbach has made the following modification of Gmelin's test. White blotting paper is dipped into the urine, and the excess allowed to drip off; it is then spread upon a white plate, and touched with a glass rod which has previously been dipped in impure nitric acid. Very distinct rings of color (yellow, violet-brown, and a

peripheral green) form at the point of contact. In a number of cases, I have noticed that Marechal's test alone gave positive results. It is well known that Gmelin's test particularly gives negative results, in febrile conditions. Frerichs found that some samples of icteric urine furnished no reaction unless exposed to the air for a certain length of time. On the other hand, too long exposure may cause the disappearance of Gmelin's reaction, because the bile pigment undergoes further changes.

The biliary acids are not always found in the urine owing to the fact that they are occasionally changed very rapidly in the blood. They are readily recognized by means of Pettenkofer's test; their aqueous solution assumes a purple-violet color on the addition of a solution of cane sugar, and concentrated sulphuric acid, if it is not heated above 70° C.

Strassburger's modification of this test is readily made. A piece of cane sugar is dissolved in the urine, a piece of filtering paper dipped therein, and then dried. If the dried paper is then touched with a glass rod which has been dipped in pure concentrated sulphuric acid, a distinct carmine or purple-violet spot forms at the end of a few minutes at the point of contact.

If the passage of bile into the intestines is entirely abolished, the feces assume an ashen gray color. At the same time the evacuations are retarded and the feces dry. They have a nauseous odor, and not infrequently contain unusual amounts of fat. In some cases, true steatorrhea, *i. e.*, the discharge mainly of masses of fat, has been observed. These changes are readily explained by the fact that the bile pigment colors the feces, that the bile stimulates peristalsis and exercises an anti-fermentative action on the contents of the intestines, and that it aids the absorption of fat.

If the icterus is the result of intra-hepatic causes, the stools are not entirely destitute of bile. They usually have a clayey appearance, but occasionally bile-stained stools alternate with unstained evacuations.

Gerhardt often observed numerous needle-shaped crystals of magnesia soaps in the feces.

The interference with the functions of the liver exercises injurious influences upon a number of other organs. Changes are observed most constantly in the digestive apparatus. The tongue usually presents a whitish, grayish, yellow or brownish coating. There is often fœtor ex ore. The patients complain not infrequently of a bitter taste in the mouth, and eructations and vomiting may be observed. As a rule, there is marked anorexia; in rarer cases, the appetite is unchanged or bulimia is noticeable. There is often a feeling of distention of the stomach, and frequent complaints of borborygmus and colicky pains.

In many patients there is a feeling of tension and tenderness in the region of the liver. The organ increases in size after a time, but later this symptom disappears while an increase of consistence becomes noticeable. If the obstruction is situated in the ductus choledochus, the gall-bladder is unusually distended with bile. It appears under the abdominal walls as a round, smooth tumor, which moves with respiration or, in other cases, is only accessible to the palpating finger. Increase in the size of the gall-bladder is also observed in occlusion of the cystic duct. Indeed, the organ may then attain the dimensions of a child's head. In such cases, the bile, which was originally present in the gall-bladder, is absorbed, and the organ becomes filled with a colorless secretion which contains mucus (*hydrops cystidis felleæ*).

The pulse is sometimes extremely slow (twenty-one beats a minute in a case observed by Frerichs). In my own experience, however, this symptom is by no means constant. It has been attributed to irritation of the intra-cardiac ganglia by the biliary acids in the blood. In cases of fever, the pulse is not accelerated in proportion to the increase of the bodily temperature.

Changes in the bodily temperature are not the result of jaundice itself.

The disposition of the patients changes very early. They become moody, capricious, and unusually irritable. Many complain of a constant insomnia.

Yellow vision (*xanthopsia*) is observed in rare cases. This must be regarded as a purely nervous symptom; in the first place, because it does not correspond to the intensity of the jaundice, and, furthermore, because it is usually intermittent. Day-blindness (*nyctalopia*) has been observed in some cases. Other writers have noticed night-blindness (*hemeralopia*). Bamberger is inclined to attach an unfavorable significance to these symptoms. Retinal hemorrhages have been repeatedly observed on ophthalmoscopic examination.

Signs of emaciation and feebleness not infrequently develop with surprising rapidity. Apart from disturbance of the functions of the liver, these symptoms are partly the result of a diminished supply of food and imperfect digestion.

It has been assumed by some that the biliary acids which are absorbed by the blood partly dissolve the red blood-globules, but these acids are in part very rapidly destroyed in the blood, and, on the other hand, are soon excreted in the urine, so that they do not attain the degree of concentration necessary to dissolve the red blood-globules. This disposes of the theory that every obstruction jaundice is associated with hæmatogenous jaundice, in which the coloring matter of the red blood-globules, which are supposed to be dissolved, is converted within the vessels into bile pigment. Leucin and tyrosin and an unusual amount of fat have been repeatedly observed in the blood.

The bile pigment is sometimes excreted in the milk of nursing women, and during pregnancy the fœtus becomes jaundiced if the icterus of the mother has lasted more than two weeks.

The duration of obstruction-jaundice depends upon its causes. In some cases the disease lasts a few days, in others a few months, and even years. As a rule, the icterus which lasts more than one to two months, or even increases in intensity, is usually due to something more serious than gall-stones or catarrh of the biliary passages. Van Swieten reports a case, however, in which recovery occurred after the jaundice had lasted eleven years. The occurrence of recovery is first recognized by the bile-staining of the fæces. A tumor of the gall-bladder, which had previously existed, disappears. When the biliary passages are suddenly freed from obstruction, bile may enter the intestines in such large quantities that thin blackish-green stools are evacuated. Next, the urine loses its dark color, and the bile-pigment reaction becomes more and more distinct. The return of the normal color appears last in the skin, since this requires a gradual desquamation of the deeper cell layers of the rete Malpighi.

If recovery does not occur, the jaundice itself may prove a source of danger. Death may occur with symptoms of increasing marasmus. Emaciation and loss of power increase, and are followed by hemorrhages

upon the skin and mucous membranes, uncontrollable diarrhœa, gastric and intestinal hemorrhages.

In other cases, severe nervous symptoms develop (cholæmia). The patients usually become apathetic, and delirium ensues; they toss restlessly to and fro, moan, and breathe irregularly. Cheyne-Stokes respiration is observed in some cases. Finally death ensues. The delirium rarely assumes a furibund character.

Death sometimes occurs suddenly, with symptoms of perforation-peritonitis, as the result of rupture of the distended bile-ducts. In other cases, rupture of the biliary canals takes place within the liver. The signs of hepatic abscess then develop, and finally prove fatal.

III. ANATOMICAL CHANGES.—A yellow color is observed in the skin, conjunctiva, subcutaneous adipose tissue, the fat about the heart and on the mesentery, and in the muscles of the body. Effusions into the serous cavities have a yellowish-brown color. Icterus has also been observed in the fasciæ, cartilages, bones, intima of the vessels, aqueous humor, occasionally in the cornea and sclerotica. Icterus is always absent in the nerve tissues, although if considerable œdema of the brain develops as the result of infiltration with serum of an icteric color, a section of the brain may present a yellowish appearance. In the liver, the biliary passages above the stenosis are dilated. If the obstruction is situated at the extremity of the ductus choledochus, all the bile-ducts may attain the circumference of the finger. At the same time, they appear elongated and sinuous. Similar changes are observed if the bile-ducts are followed into the parenchyma of the liver; indeed, sacculated diverticula are here aggregated still more closely than in the ducts. In some portions of the surface of the liver they project like cysts, which are filled with biliary contents. The gall-bladder also takes part in general dilatation of the bile-ducts.

The walls of the bile-ducts are often thickened, in other cases, extremely thin. At first, their contents consist of unchanged bile, but if the obstruction persists for a long time, and the liver loses its bile-producing function, the bile is absorbed and replaced by a more serous, mucous fluid, which is furnished by the mucous membrane of the biliary passages.

At the beginning of general biliary stasis, the liver increases in size, but later it becomes smaller. Its consistence often changes; sometimes it is unusually flabby, sometimes extremely hard and firm. In recent cases, its color is golden or saffron-yellow; in older ones, greenish or greenish-black. The immediate vicinity of the central veins is most intensely colored, so that the liver not infrequently presents a nutmeg appearance.

Under the microscope, the hepatic cells, particularly near the central veins, are in part diffusely infiltrated with bile, in part filled with granular bile pigment. This first accumulates near the nuclei of the cells, sometimes in the shape of fine granules, sometimes as irregular jagged dots, sometimes as small delicate rods. Later, some of the hepatic cells undergo granular degeneration; not infrequently the interlobular connective tissue undergoes proliferation (cirrhosis). The interlobular biliary passages are not infrequently filled with thickened, blackish-green bile. Frerichs noticed that the cylindrical epithelium is gradually flattened, and assumes more and more the appearance of pavement epithelium. Fat granules are observed here and there in these cells.

After prolonged obstruction jaundice, important anatomical changes

develop in the kidneys. Externally they appear greenish; in old cases, the cut section is also greenish; in recent cases it presents an intense saffron color. The consistence is often extremely soft and flabby.

At first, the epithelium cells of the convoluted tubes and Henle's loops are diffusely covered with bile pigment; later they contain a deposit of granular bile pigment. The yellow color is least intense in the Malpighian capsules. At a still later period, there is a deposit of granular pigment in the lumen of the tubes, in which we find pigment casts, especially in the straight tubes of the pyramids. Under such circumstances, flakes, granules, and cylindrical casts of bile pigment are found in the sediment of the urine. These lesions may obstruct the renal tubules, and thus render impossible the excretion of the constituents of the bile which have been taken up from the blood. The development of cholæmic symptoms is favored in this manner. In addition, fatty degeneration, desquamation, and destruction of epithelium of the tubes are sometimes observed.

IV. DIAGNOSIS.—The diagnosis of obstruction jaundice is easy, but it should be remembered that daylight is requisite to recognize the icteric color of the skin. In anæmic and cachectic individuals, we should avoid mistaking an excessive development of subconjunctival adipose tissue for conjunctival jaundice. It is not always easy to distinguish obstruction jaundice from hæmatogenous icterus, *i. e.*, that form which develops from the dissolution of red blood-globules within the vessels and their conversion into bile pigment. In the latter, biliary acids will always be absent in the urine; but, on the other hand, they cannot always be demonstrated in this fluid in undoubted cases of obstruction jaundice.

On the other hand, biliary acids have been detected in the urine of healthy individuals. In addition, the stools retain the normal color in hæmatogenous icterus, since the flow of bile has not been interrupted; bile-stained stools may also occur in obstruction jaundice, if it is the result of intra-hepatic causes, and not of complete closure of the ductus hepaticus or choledochus. In order to determine whether an obstruction jaundice is the result of diseases of the hepatic parenchyma or of causes situated outside of the liver, careful examination should be made of all the organs in question, and, in addition, attention should be paid to the character of the stools. These are rarely constantly destitute of bile when the jaundice is the result of intra-hepatic causes. It is only in affections of the ductus choledochus that an increase in the size of the gall-bladder may be expected.

V. PROGNOSIS.—The prognosis depends upon the causes in each individual case. It is unfavorable when symptoms of dissolution of the blood occur, since these indicate an advanced stage of marasmus. Cholæmic symptoms also have an unfavorable significance.

VI. TREATMENT.—The chief importance must be attached to the diet; fats must be particularly avoided. We may recommend skimmed milk, coffee, tea, soup, lean meat, stewed vegetables, and fruit, soft boiled eggs, and white wine. If the thirst is increased, we may give lemonade or water, Selters water, etc.

If the patients complain of a bitter taste in the mouth, the tongue is coated, and nausea is present, we may order acids, for example: \mathcal{R} Sol. acid. hydrochloric., \mathfrak{M} xij. : \mathfrak{z} vi. D. S. One tablespoonful every three hours. \mathcal{R} Acid. hydrochloric., acid. nitric., $\mathfrak{a}\mathfrak{a}$ \mathfrak{M} viiij.; aq. destil. ad \mathfrak{z} viiss. M. D. S. One tablespoonful every three hours, etc.

Constipation necessitates the administration of laxatives, especially the vegetable ones (℞ Inf. rad. rhei, ʒ ss. : ʒ vi. One tablespoonful every three hours. ℞ Inf. sennæ comp., ʒ v. One tablespoonful t. i. d. ℞ Pulv. liq. comp., ʒ i. D. S. One to two teaspoonfuls at night. ℞ Aloes, ext. rhei comp., āā gr. xxij., pulv. et succ. liq., q. s. ut ft. pil. No. xxx., three to four pills at night). Calomel may be given in a single dose of gr. iv.-vij.

If organic changes in the liver can be excluded, and we have to deal merely with chronic absorption jaundice, we may make use of mineral waters. If the jaundice is associated with obstinate jaundice, we may order waters containing Glauber's salts and sodium chloride—in plethoric individuals Marienbad, Kissingen or Homburg, in feeble individuals Carlsbad, Soden or Kronthal. In addition, the alkaline and alkaline-muriatic wells, such as Ems, Vichy, Selters, Bilin, etc.

When necessary, diuresis should be stimulated by the administration of carbonated waters (for example, Selters), acetate or bitartrate of potash, etc.

I have relieved several cases of annoying pruritus by large doses of potassium bromide (gr. xxx. morning and evening). The surface may also be washed with a solution of carbolic acid (2%), corrosive sublimate, vinegar, warm baths, etc.

Warm baths are agreeable to the majority of patients. Their efficacy is increased by the addition of about fifteen ounces of soda to each bath.

In my experience, chloral hydrate (gr. xxx. to xlv. in a wineglassful of sugar-water) gives the best results in obstinate insomnia.

The development of cholæmic symptoms requires stimulant measures, for example: ℞ Acid. benzoic., gr. ivss.; camphor., gr. i.; sacch. albi, gr. viiss. M. f. p. No. x. S. One powder every two hours; strong wine, mustard poultices to the nape of the neck, ice-bag to the head, cups to the neck, etc.

2. *Catarrh of the Bile-ducts. Cholangitis et Cholecystitis Catarrhalis.*

(*Catarrhal or Simple Jaundice. Gastro-duodenal Jaundice.*)

I. ETIOLOGY.—Catarrh of the bile-ducts occurs in rare cases as a primary affection following a cold. It is almost always the result of secondary changes, most frequently of the extension of inflammation to the ductus choledochus and often higher up from previous gastro-duodenal catarrh (vide page 105).

Concretions or parasites in the bile-ducts sometimes give rise to catarrh of the mucous membrane by their purely mechanical irritation. According to some authors, this catarrh may be the result of anomalies in the chemical constitution of the bile. According to Frerichs, prolonged fasting causes stasis of bile within the gall-bladder, favors decomposition of the accumulated bile, and may thus produce catarrh which is confined to the gall-bladder and cystic duct.

Catarrh of the intra-hepatic biliary canals is not infrequently produced by diseases of the hepatic parenchyma (cancer, abscess, echinococcus, and cirrhosis). The diagnosis of catarrh, instead of compression of the biliary passages, may be made in these conditions, if jaundice occurs transitorily despite the continued advance of the affections mentioned.

The disease is sometimes the result of disturbance of circulation

(valvular disease of the heart, emphysema of the lungs, chronic bronchitis, and other chronic diseases of the respiratory tract).

Cases have been described in which signs of catarrh of the bile-ducts repeatedly occurred during menstruation, probably as the result of vicarious congestion of the liver.

In those cases in which the symptoms follow mental excitement, we are inclined to attribute the disease to vaso-motor disturbances, and anomalies in the distribution of the blood to the liver. Toxic catarrh of the bile-ducts may be the result of poisoning with phosphorus, lead, and chloral hydrate. This category also probably includes those forms which are observed during infectious disease (fibrinous pneumonia, typhoid fever, cholera, malaria, and the beginning of the so-called secondary stage of syphilis).

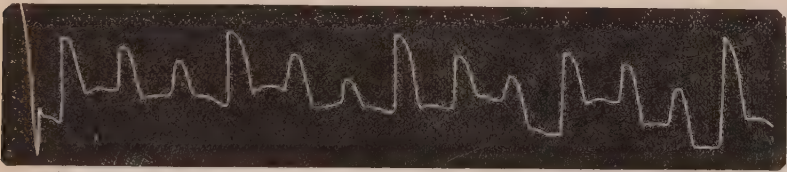
The epidemic occurrence of catarrh of the bile-ducts has been described by a number of writers. In some cases it is the result of colds, and hence is observed during periods of marked changes of temperature. Koehnhorn attributes some epidemics which have occurred in barracks to the monotony of the diet.

Certain epidemics are undoubtedly the result of miasmatic influences.

Rehn described an epidemic in Hanau in which children alone were affected; in other cases, adults alone suffered from the disease.

Catarrh of the bile-ducts occurs with equal frequency in both sexes. The disease is rare in old age.

FIG. 49.



Pulsus trigeminus in catarrhal jaundice in a man æt. 32 years; right brachial artery.

II. SYMPTOMS.—Catarrh of the bile-ducts cannot be recognized unless occlusion, and therefore stasis of bile, takes place as the result of swelling of the inflamed mucous membrane, excessive secretion of mucus, and active desquamation of epithelium. The symptoms are then the same as those of the obstruction jaundice just described, so that we need not again enter upon their description. It may here be mentioned, however, that slowness of the pulse is especially frequent in catarrhal jaundice, and that allorhythm of the pulse is not infrequently observed (vide Fig. 49).

As a rule, the occurrence of jaundice is preceded by the symptoms of gastro-duodenal catarrh (coated tongue, flat taste in the mouth, anorexia, eructations, nausea, vomiting, tenderness in the gastric region, etc.) Febrile movement is observed not infrequently. These prodromata sometimes last two weeks, but in some cases they are so slight as to be almost overlooked. After the appearance of jaundice the gastro-enteric symptoms sometimes diminish entirely; in other cases they increase in severity. The duration of the jaundice, on the average, is two to six weeks, but under certain circumstances it may last for several months. The disease almost always ends in recovery. In rare cases, cholæmic symptoms develop and terminate fatally. Adhesion of the bile-ducts sometimes occurs in those places at which active desquamation of the epithelium has occurred, and this results in chronic jaundice. In equally

rare cases, the catarrh is followed by ulceration and hepatic abscess. Catarrh of the bile-ducts may also give rise to the formation of gall-stones.

III. ANATOMICAL CHANGES.—The changes in this disease can rarely be observed in the dead body. In addition, a part of the symptoms (congestion and swelling of the mucous membrane) disappear in the cadaver.

Nevertheless, we are justified in assuming congestion, swelling, and unusual succulence of the mucous membrane in this condition. An accumulation of mucus and desquamated epithelium cells remains in the ducts as signs of a previous inflammation. Occasionally we find that their consistence is increased, so that they float to and fro in water. In older cases, they are not infrequently of a puriform character. Stenosis and occlusion are especially apt to occur at the entrance of the ductus choledochus into the duodenum. Pressure along this duct in the direction towards the intestine often discharges a plug of mucus and epithelium. In other cases, the stenosis is rather the result of swelling of the mucous membrane which has disappeared in the cadaver, so that the mechanical conditions are explained with difficulty. Under such circumstances, it should be remembered that the mucous membrane is pale in that portion of the duct which was occluded, while it appears yellow above on account of the stasis of bile. The biliary passages are dilated and filled with bile above the site of occlusion. If the catarrh was confined chiefly to the gall-bladder, its contents contain an unusual amount of mucus. This inflammation is sometimes followed by thickening or calcification of the wall of the gall-bladder, adhesion, inflammation of the serous surface, the formation of concretions, or dropsy of the organ. The liver is icteric, and may present the changes described in the previous section.

IV. DIAGNOSIS.—The recognition of catarrh of the bile-ducts is generally easy if obstruction jaundice is present. Careful examination of the liver and adjacent organs and an accurate history of the case will generally enable us without special difficulty to determine the cause of the disease.

We should be careful, however, not to make too positive a diagnosis. We recently had under treatment a man, *æt.* 75 years, who suddenly suffered from constipation and jaundice, presumably catarrhal in character. At first the liver appeared unchanged. Three weeks later, it increased in size; at the end of another month, prominences appeared upon its lower surface, and in the thirteenth week death occurred from cancerous cachexia. Our suspicions should always be aroused if jaundice occurs in old age, and its intensity does not diminish at the end of four weeks.

Catarrhal jaundice may be mistaken for gall-stones which have obstructed the ducts; but in the latter, colicky pains are usually present, the jaundice generally disappears more rapidly, and finally the gall-stones should be looked for in the stools.

V. PROGNOSIS.—This is almost always favorable.

VI. TREATMENT.—The causal indications should be first considered. If the patient suffers from gastro-duodenitis, he should be placed on fluid diet (milk, soup, tea, lemonade) and acids given internally (acid hydrochloric, \mathfrak{M} xxx. : $\frac{z}{3}$ viiss., one tablespoonful every two hours, etc.). If the stomach is overloaded, an emetic may be ordered. The vigorous contractions of the abdominal walls sometimes dislodge the plug in the

bile-ducts. These remedies, however, should be used with caution, since it is known that they not infrequently give rise to jaundice.

Gerhardt recommended compression and faradization of the gall-bladder in order to remove, mechanically, the plug in the bile-duct.

This writer found that, in compression of the gall-bladder, the entrance of bile into the duodenum could sometimes be felt as a fine râle. The stools then usually assume a biliary color on the second day; the appetite improves at once, and the pruritus which is produced by jaundice also ceases.

In faradization of the gall-bladder Gerhardt applies one pole to the region of the organ, the other posteriorly along the spine. A strong current should be used. This method should be employed when the gall-bladder cannot be reached with the fingers. If the application is successful, the urine soon becomes lighter in color and its specific gravity diminishes.

Krull has repeatedly obtained very rapid results from the infusion of large amounts of water (one to two litres at 12° R.; later, at 18°) into the large intestine. The patient should endeavor to retain the water as long as possible.

3. *Purulent Inflammation of the Bile-ducts.*

Suppurative Cholangitis and Cholecystitis.

I. This is observed most frequently in gall-stones, parasites, or foreign bodies in the bile-ducts. The inflammation in the beginning is generally catarrhal, then becomes purulent, and, in some cases, even diphtheritic and croupous. The suppurative inflammation is sometimes the result of stasis of bile, the latter undergoing decomposition and irritating the mucous membrane to an intense degree. In other cases the inflammation extends from the liver (abscess, inflammation of branches of the portal vein, etc.)

Purulent inflammation sometimes develops from catarrh of the bile-duct. In some cases the disease is the result of infectious diseases (typhoid fever, pyæmia, etc.). Cases have also been reported in which no cause could be discovered.

II. The inflammation is sometimes confined to the gall-bladder, sometimes to the excretory ducts, sometimes to the intra-hepatic canals, or finally, it may extend over the entire system of the biliary passages. The contents of the biliary passages contain an admixture of pus, sometimes they are almost entirely purulent, occasionally ichorous. The mucous membrane is loosened, injected, sometimes ecchymotic, or if the condition has lasted for a long time, it is pigmented in spots. Losses of substance may occur and give rise to perforation either into the abdominal cavity or, after peritonitic adhesions have formed, into the stomach, duodenum, colon, portal vein, external abdominal walls, etc. In purulent inflammation of the intra-hepatic ducts, the inflammation sometimes extends to the parenchyma, inasmuch as ulcerations of the canals cause partial destruction of the walls, and thus inflammation and abscess formation in adjacent parts of the liver. The abscesses are generally multiple and small in size, more rarely they are single and attain large dimensions.

III. A diagnosis can hardly be made with certainty during life. All symptoms may be absent, particularly in infectious diseases in which the primary disease occupies the foreground. Jaundice, chill, fever, enlargement of the liver, and perhaps painful swelling of the gall-bladder are occasionally observed. The disease sometimes ends fatally in a few days, or it is more protracted and ends in recovery. For example, gall-stones may be discharged and the retained masses of pus may then find their way into the intestines. The prognosis is always grave. The treatment is symptomatic.

4. *Gall-Stones. Cholelithiasis.*

I. **ETIOLOGY.**—The disease is observed most frequently in women after the age of forty years. Among three hundred and ninety-five cases, Hein found only three below the age of twenty years.

In a few cases, however, the calculi have been found in still-born children, infants, and during the first decade. Compared with men, women are affected in the proportion of 3:4-1. The predisposition of females has been attributed to right living, which interferes with the flow of bile, and to the relations between the liver and the sexual organs of the female. It has been noticed that the development of gall-stones is favored by previous pregnancy, and it develops most frequently at the menopause, or that the first symptoms become noticeable at the beginning of the period of puerity.

According to some writers, solitary helminths predispose to the development of gall-stones. Summary statements are made with regard to high living. Local and climatic influences are considered important by some writers. In some regions this has been attributed to the abundance of lime in the drinking-water. Gall-stones are extremely rare in the regions in which abscess of the liver and hepatic enlargement are usually frequent. Benceke has called attention to the fact that the disease is often associated with enlargement of the arteries. In some cases the gall-stones form around foreign bodies, such as casts of blood, parasites, etc. Mettenheimer believed that fatty and calcified cells of the mucous membrane of the gall-bladder, when they had been separated from the latter, may become the nuclei of the gall-stones.

III. ANATOMICAL CHANGES.—Gall-stones form most frequently in the gall-bladder, much more rarely in the intra-hepatic ducts, while they are never found in the ductus cysticus, hepaticus, cholecysticus, unless they have migrated from other parts. Bile gravel is a finely granular mass, which sometimes lacks incrustated, it is usually greenish, blackish, more rarely brown, or even whitish in color. It is sometimes found alone, sometimes in combination with gall-stones. It is most frequent in the gall-bladder, in which its development is favored by the physiological stagnation of the bile.

The size of gall-stones varies from that of a grain of sand to that of a hen's egg. Those situated in the gall-bladder are usually larger than those found in the narrow intra-hepatic ducts. Merkel described one which was fifteen cm. in length and twelve cm. in circumference. Their number is extremely variable. Sometimes but a single one is found, in other cases they are almost innumerable.

On the average, the gall-bladder contains ten to fifteen, in one case one counted 86 in thousand eight hundred and two. Choport reports a case in which the intra-hepatic ducts were distended with gall-stones to such an extent that the liver could hardly be cut with a scalpel.

The shape of the stones depends partly on the place of development. In the intra-hepatic ducts they are often cylindrical, not infrequently with lateral projections just at the branches of the ducts. Such gall-stones are sometimes hollow, and the hollow may be filled with incrustated bile. Stones in the gall-bladder may be round, elongated, or angular. Sometimes they resemble the pear shape of the gall-bladder, especially if only one stone is present which completely fills the organ. In not a few cases the shape resembles that of a nutmeg. Sometimes we find regular stereometric bodies, for example, tetrahedra, octahedra, etc. More frequently, the calculi are irregular in shape, and their surfaces often fit into one another (vide Fig. 50).

It was formerly supposed that the many angles of the gall-stones were the result of the friction of the calculi against one another; but this view is incorrect. Their shape is at first round, and spaces are thus left between adjacent stones. Hence, the farther growth of the calculi must deviate from the spherical shape.

The surface the calculus may be entirely smooth. In other cases it presents numerous nodules (mulberry stones).

The color may be black, greenish-brown, white, yellow, or gray, and depends particularly upon their chemical constitution. Calculi

FIG. 50.

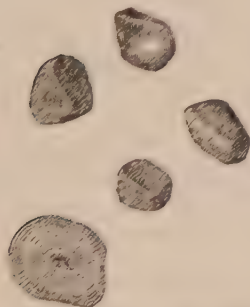


FIG. 51.

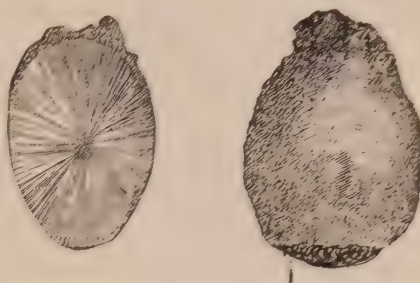


FIG. 50.—Facetted gall-stones: the one to the left has been sawn through. Natural size.
FIG. 51.—Gall-stones of cholestearin: the one to the left has been sawn through. Natural size.

composed of cholestearin are white as alabaster, appear transparent when fresh, and only lose this appearance after they have been exposed to the air. The brownish, greenish, or blackish color becomes more marked the greater the amount of bile pigment which is present. Calculi of calcium carbonate have a chalky whiteness. In very rare cases, bluish gall-stones have been observed. In the fresh condition, they can usually be compressed between the fingers. Their surfaces often have a

FIG. 52.



FIG. 53.

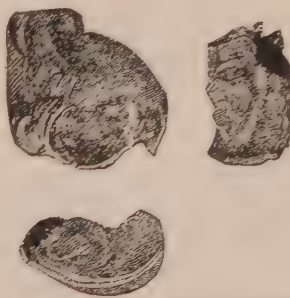


FIG. 52.—Cholestearin calculus with cortex of bilirubin-lime. On the right side the surface has been sawn through. Natural size.
FIG. 53.—Laminated calculus of bilirubin-lime. This stone was crushed during evacuation.

peculiar fatty or soapy feel. Fragments of the calculi are occasionally found in the gall-bladder or excretory ducts. The surface of fracture of cholestearin calculi is radiating and crystalline (vide Fig. 51). In those which are composed chiefly of bilirubin-lime, the surface of fracture is often very irregular; more rarely the stone consists of concentric layers (vide Fig. 53). The weight depends upon the size and chemical constitution. Those formed of cholestearin are especially

light. Ritter reports a case in which a single gall-stone weighed 4½ ounces. The specific gravity is always greater than that of water, in one case it was 1.580, in another 1.966. After the calculi have been exposed to the air for a long time, and have become desiccated, they may float on water. After they have re-absorbed water they again sink.

The following substances have been found in gall-stones: Cholestearin, bile-pigments, especially bilirubin, which, in combination with lime, is found in most calculi, smaller quantities of the biliary acids, fatty acids, mucin, and epithelium. The inorganic substances include carbonate, phosphate, and sulphate of lime, phosphate and carbonate of magnesia, traces of iron, copper, and manganese. Mercury is sometimes found if the patients have been subjected to mercurial inunctions.

In some gall-stones, cholestearin is the chief constituent (ninety-eight per cent), and it is only in rare cases that this substance is entirely absent. Ritter found, as the result of numerous analyses, that the majority of the calculi contained:

Cholestearin,	70.6
Organic substances,	22.9
Inorganic substances,	6.5

Ritter also furnishes the following detailed analysis of a gall-stone:

Cholestearin,	0.4
Bilirubin and bilifuscin,	0.6
Biliprasin,	0.8
Bilihumin,	12.8
Biliary constituents, soluble in water,	2.3
Calcium carbonate,	64.6
Calcium phosphate,	12.3
Ammonium-magnesia phosphate,	3.4
Mucus,	2.8

Biliary calculi are either homogeneous or conglomerate. The former have the same physical and chemical constitution throughout. In the centre of the latter we find a nucleus and the peripheral layers often present a special cortex. Thus Fig. 51 represents a cholestearin calculus, with a simple nucleus, while in Fig. 52 we notice a nucleus, a body composed of cholestearin, and a cortex composed, in the main, of bilirubin-lime.

The nucleus of gall-stones usually consists of mucin, epithelium cells, and bilirubin-lime, in some cases of foreign bodies. The nucleus is sometimes situated eccentrically. This is always the case when more than one is present (as many as five have been observed). Multinuclear gall-stones usually result from the conglomeration of single ones.

With regard to their chemical constitution, we may distinguish: *a.* pure cholestearin stones; *b.* pure pigment stones; *c.* pure lime stones; *d.* cholestearin pigment stones.

a. Pure cholestearin calculi are transparent in transmitted light when in a fresh condition; but when dry, become opaque. They usually have a crystalline, glistening surface of fracture, and a radiating structure. The surface is sometimes smooth, sometimes nodular. They have a peculiar fatty structure, and are usually light. The specific gravity is also very low, though always higher than that of water. They are combustible, and burn with a bright flame. *b.* Pure pigment stones are much rarer than cholestearin calculi. They may be of a rusty brown, greenish or blackish color, are often homogeneous and not infrequently

very brittle. *c.* Pure lime stones (composed of carbonate of lime) are very rare. They are white or grayish-white, very hard and heavy. *d.* Cholestearin pigment stones form the most frequent variety. Their structure is not infrequently laminated, or the body consists chiefly of cholestearin, the cortex contains bilirubin-lime or carbonate of lime. If a number of calculi are contained in the gall-bladder, they almost always present the same physical constitution.

The surface of gall-stones is sometimes eroded and more or less destroyed (vide Fig. 54). It has been supposed, therefore, that the bile itself is capable of dissolving gall-stones. Schueppel has recently suggested that this is the result of the proliferation of bacteria.

The calculi often exert a noxious influence on the integrity of the biliary passages. They sometimes distend the gall-bladder to such an extent that it is unable to receive fluid bile. In other cases, it contains also a mucous or purulent fluid and indications of catarrhal inflammation of the mucous membrane. This fluid is sometimes converted into a mortar-like mass. The mucous membrane is sometimes very smooth, like a serous membrane; sometimes its normal recesses are distended into diverticula, which may also contain gall-stones; the latter may become encapsulated. Fatty degeneration of the muscular coat, calcification, hypertrophy, and atrophy of the walls are not infrequent.

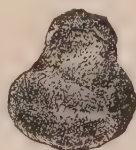
Similar anatomical changes may occur in the intra-hepatic ducts. The parenchyma of the liver itself may be implicated, and hepatic abscess, the formation of cicatrices, or inflammation of the portal vein may be the result.

The real causes of the formation of biliary calculi are not known with certainty. In the majority of cases, it seems that their development is preceded by catarrh of the biliary passages. The abundant mucus which is thus produced decomposes the bile, particularly the salts of the biliary acids. Inasmuch as the biliary acids retain cholestearin and bile pigment in solution, changes in the former may render possible the deposit of the hitherto dissolved substances. It is also probable that the carbonate of lime is derived from deposits in the bile, although it is possibly at times a direct product of the inflamed mucous membrane. In a number of cases, the formation of gall-stones has been observed in the glands of the mucous membrane.

III. SYMPTOMS.—In many cases, the presence of gall-stones remains entirely latent during life. In others, they give rise to no annoyance, but can be positively diagnosed, for example, if the gall-bladder is distended with calculi, and can be felt as a hard, tense tumor. If the gall-bladder contains several stones, we are sometimes able to rub these against one another, thus giving rise to a peculiar hard friction. Upon the application of the stethoscope, this is heard as a metallic clattering. In a case in which it was doubtful whether the enlargement of the gall-bladder was due to cancer or to an accumulation of gall-stones, Whitaker employed puncture, and attempted to base the diagnosis of gall-stones upon the hard scratching of the instrument employed in puncture.

Gall-stones are sometimes found accidentally in the fæces, although they have given rise to no symptoms. This is observed in old people in whom the symptoms remain absent on account of flaccidity and unusual dilatation of the biliary passages and the diminished sensibility of the mucous membrane.

FIG. 54.



Bile pigment calculus with numerous erosions and depressions.

In some cases, the calculi are concealed by the symptoms of other diseases, for example, inflammatory changes in the intra-hepatic ducts, and abscess of the liver.

The typical clinical history by which the presence of gall-stones is revealed is that of biliary colic. But this occurs only when the gall-stones have a tendency to migrate, and meet with obstructions in their passage. The attacks of colic sometimes occur spontaneously, sometimes follow bodily exertion, occasionally mental emotion. As a rule, the colic appears some time after a hearty meal. The most prominent symptom is the atrocious pain, of a boring, burning, lancinating character, and situated in the right hypochondrium. It generally radiates into the right thigh and testicle, the back, the right, more rarely the left, shoulder, and the right arm. The face expresses pain and terror, the forehead is covered with perspiration, and the features are pale, or, in rare cases, unusually red. The patients often toss to and fro in bed, press the fists into the region of the liver, and are bent over forwards. The abdominal walls are tense and contracted; sometimes this is found only on the right side. The region of the liver is unusually sensitive to pressure, and in many cases the patients cry out aloud, as soon as the region of the gall-bladder is touched (outer border of the right rectus abdominis, immediately below the eighth rib).

A chill occurs not infrequently at the beginning of the attack. The temperature may gradually rise beyond 40° C. In other cases, it remains normal, or even subnormal. The pulse is not infrequently intermittent or irregular, sometimes rapid and small, sometimes slow and full; the respirations are interrupted and deep.

In many cases, vomiting occurs, at first of the contents of the stomach, then of bilious, and finally of muco-watery masses. Obstinate singultus is not an infrequent symptom. The bowels are generally constipated, but in some cases diarrhoea is noticed (muco-bloody or rice-water discharges). The urine is scanty, dark, and loaded with urates.

The violent pains occasionally cause severe disturbance of consciousness. The patients become delirious, and general convulsions and unconsciousness may be produced. As a rule, the average duration of an attack of pain is three to five hours; then a pause follows, after which the pains again occur. Finally they may cease quite suddenly.

The pains must be attributed to the irritation of the mucous membrane of the excretory ducts by the gall-stones. After the calculus has left the ductus cysticus, the pains generally cease for some time, because the ductus choledochus is much wider than the former. The pains reappear as soon as the stone passes through the narrow outlet into the intestine. The calculi of the intra-hepatic canals generally pass along without special mechanical difficulty, and therefore without pain until they reach the point of outlet of the ductus choledochus. An attack of biliary colic not infrequently ceases because the gall-stone which has passed from the gall-bladder into the ductus cysticus is again pushed into the former.

In not a few cases, the symptoms mentioned are associated with jaundice; sometimes only the conjunctiva is affected, sometimes the skin and urine.

The symptoms vary according as the calculus, when it passes the ductus hepaticus or choledochus, merely slows or entirely abolishes the flow of bile. Whether the gall-stone is present in the ductus hepaticus or choledochus may be determined by the fact that in the latter event the

gall-bladder increases in size, and becomes accessible to palpation. The icterus develops some time after the beginning of biliary colic, since stasis of bile and the accumulation of bile pigment in the blood must attain a certain intensity before distinct jaundice is produced.

The evacuations should be dissolved under a vigorous stream of water in a coarse sieve, so that the latter may retain any calculi which have entered the intestine. The stools should be examined daily for a week after the beginning of an attack, since the calculi are often retained in the intestine for a number of days.

In the majority of cases biliary colic terminates in recovery, but there is danger of relapse, particularly if we find only a single faceted calculus in the stools. In rare cases, violent biliary colic may result in death. The intensity of the pain may give rise to rapid collapse, and the patients die, as in shock, from paralysis of the heart. Hemorrhage into the brain has been observed occasionally in old people during an attack of biliary colic.

Incarceration of the gall-stone in the biliary passages occasionally occurs. This is followed by the symptoms of total obstruction of the bile-ducts; marked jaundice, enlargement of the liver, later atrophy of the liver, death with cholæmic symptoms, or, if the ductus cysticus is alone occluded, hydrops cystidis felleæ.

In some cases this is preceded by rupture of the bile-ducts with sudden symptoms of exhaustion and perforation-peritonitis to which the patients rapidly succumb.

In other cases, the incarcerated gall-stones give rise to inflammatory and ulcerative processes. The gall-bladder or bile-ducts become adherent to adjacent organs, enter into communication with them, and empty into them their calculous contents. Thus, communication has been observed with the stomach, so that in some cases the gall-stones are vomited. The fistula may also open into the transverse colon, duodenum, more rarely the ileum. As a rule, gall-stones cannot pass through the normal bile-ducts if their diameter exceeds one cm. Larger gall-stones in the stools can hardly have entered the intestinal tract except by means of abnormal communications. The formation of a fistula and the rupture of gall-stones into the kidneys and bladder have also been observed. Sometimes the gall-bladder becomes adherent to the abdominal walls, and an external fistula forms through which gall-stones and bile are discharged. Not infrequently the perforation does not occur until a sort of cavity has been developed in the vicinity of the gall-bladder by circumscribed peritonitis, and this then gives rise to the external fistula. The opening of the fistula is sometimes a considerable distance from the liver, for example, at the umbilicus, or a little above Poupart's ligament. Such conditions are sometimes well tolerated for an extremely long time. Philipson reports a case in which pregnancy and delivery occurred despite the existence of an external fistula.

If, at the same time, the ductus choledochus is occluded by a calculus, almost all the bile may escape through the external fistula. If the cystic duct is also occluded, the escaping fluid soon loses its biliary character, assumes a more colorless and mucous appearance, like that of the secretion of the mucous membrane of the gall-bladder.

Calculi in the intra-hepatic canals may also perforate through the diaphragm, lungs, and bronchi, in rare cases into the portal veins. After the gall-stones have entered the intestines, the danger is usually, though not always, past. In some cases the calculus is so large, or is

situated in such a peculiar manner, that it occludes the intestine and gives rise to the symptom of ileus. Cases have also been reported in which numerous gall-stones became adherent to one another through the agency of the fæces, and thus caused intestinal occlusion. Sometimes a gall-stone remains immediately above the anus, and must be removed by the finger; smaller ones may enter the vermiform process, and give rise to typhlitis, perityphlitis, and paratyphlitis. It may also be mentioned that the symptoms of pyloric stenosis have occasionally been observed as the result of gall-stones (compression of the pylorus by the distended bile-ducts). Obliteration of the bile-ducts may also be owing to the fact that the gall-stones, in their passage, have injured the mucous membrane, and subsequent cicatrization has given rise to adhesion of the opposite walls.

We cannot enter into all the symptoms which may be the result of biliary calculi, but will merely refer to a few changes in the liver: abscess, cirrhosis, and even cancer. Among the complications may be mentioned renal calculi (in six per cent of Kraus' cases), gout (two per cent), diabetes mellitus (four per cent), and glycosuria (seven per cent).

IV. DIAGNOSIS.—Biliary calculi may be mistaken for the following diseases: A. Gastralgia. In gastralgia, the pain is more associated with the ingestion of food; it is confined to the gastric region proper, is preceded by gastric symptoms, and jaundice is absent.

B. Colic and Lead Colic. Flatulence often present in colic, and, as a rule, the pain diminishes in intensity upon pressure over the painful spot. In lead colic, the previous history and the blue line on the gums must be taken into consideration.

C. Hepatic Neuralgia. Many writers believe that attacks of pain in the hepatic region may be the result of purely nervous causes. These alternate occasionally with neuralgia in other nerve tracts. Treatment which is directed against gall-stones is entirely useless in such cases.

D. Typhlitis, perityphlitis, and paratyphlitis. The pain is chiefly confined to the right iliac fossa, and in addition we should search for a tumor in this region.

E. Renal colic. Attention should be paid to the passage of gravel in the urine, blood in the urine, and pains in the region of the kidneys.

F. Internal incarceration. This is associated with vomiting, obstinate constipation, and vomiting of fæces.

G. Psoas abscess. Fiedler saw a case of this kind in which large gall-stones had been retained in the sigmoid flexure.

H. Aneurisms of the celiac axis or abdominal aorta. We should search for abnormal pulsation and murmurs in the vessels.

I. Intermittent fever. A mistake is possible if the pains are not severe, and chill and fever occur daily; but quinine is useless in biliary colic, and in addition, the chill and elevation of temperature in the latter usually occur, not in the morning but in the afternoon.

K. Cholera. During a cholera epidemic mistakes may arise if obstinate vomiting and watery stools occur in biliary colic.

L. Poisoning. If biliary colic proves rapidly fatal, the suspicion of poisoning may arise.

V. PROGNOSIS. As a rule, the prognosis is good, but it should be given with caution since there is a possibility of unexpected accidents. On the other hand, the hope of recovery should not be given up too soon. Frerichs observed recovery after the ductus choledochus had been

occluded several months. There is always great danger, however, of a recurrence of the attacks.

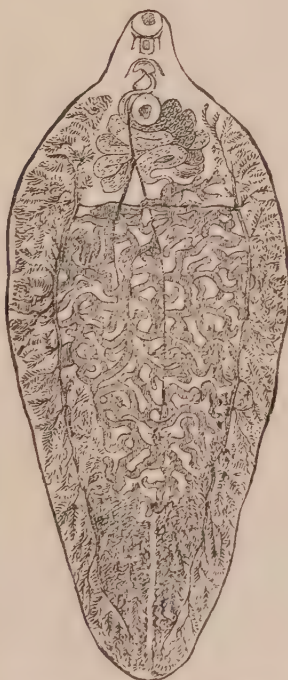
V. TREATMENT. Biliary colic should be treated by large doses of narcotics. We give the preference to chloral hydrate (gr. xlv. in a wineglass of sugar water) or morphine. In addition, rest in bed and warm poultices, no solid food, and Selters, Vichy, or Ems waters as drinks. The narcotics not alone relieve the pain, but also relax the spasmodic muscular fibres of the biliary passages. If we have reason to believe that the calculus has entered the intestine, a mild laxative may be given. The administration of emetics during an attack is very dangerous, since the biliary passages may be ruptured during the act of vomiting. In

FIG. 55.



Distomum hepaticum.
Natural size.

FIG. 56.



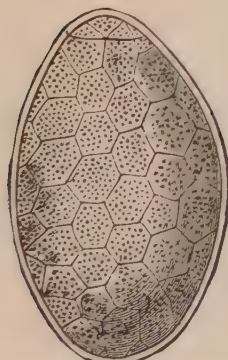
Distomum hepaticum. Enlarged
10 times. After Leuckart.

FIG. 58.



Distomum lanceolatum. En-
larged 10 times. After
Leuckart.

FIG. 57.



Ovum of *Distomum hepaticum.* Enlarged 400 times.

FIG. 59.



Ova of *Distomum lanceola-*
tum. Enlarged 400 times.

very obstinate cases it may be necessary to give chloroform for hours. Chloroform has also been given internally as a solvent. According to Kennedy, $\frac{5}{vi}$ olive oil taken internally, favors the passage of the gallstones. To prevent a relapse, the patient should live regularly, take exercise, and avoid the excessive ingestion of meats and alcoholics. I am inclined to believe that I have obtained good effects in a number of cases by the prolonged administration of salicylic acid (gr. viiss. t. i. d.). We should order alkaline wells (Carlsbad, Ems, Vichy), but the patient should be told that a violent attack of colic may occur at the springs. The cure should be repeated for several years in succession. During

the winter the patients may take alkaline acidulous waters. In cases in which attacks of biliary colic recur constantly, Langenbuch has successfully performed extirpation of the gall-bladder (cholecystotomy).

5. *Parasites of the Biliary Passages.*

The animal parasites found in the biliary passages are the *ascaris lumbricoides*, *echinococcus*, and *distomum*.

a. *Ascaris lumbricoides* is never found in the biliary passages unless it migrates from the intestines through the ductus choledochus, or abnormal communications exist between the biliary passages and the intestines. In many cases the migration occurs post-mortem, in others it happens during life, the worms passing into the gall-bladder or the larger branches of the intra-hepatic canals. They may give rise to occlusion of the bile-ducts and hepatic abscess, or may form the nucleus of gall-stones after they have died. A diagnosis is impossible, unless an abscess of the liver is discharged externally and an ascaris is discharged at the same time.

b. *Echinococci* either enter the bile-ducts from the liver (and then give rise to the symptoms of biliary colic in their passage to the intestine) or they develop within the biliary canals.

c. *Distomum hepaticum* and *distomum lanceolatum* occur most frequently in the sheep, but are found, in rare cases, in the human being. In the majority of cases they were found accidentally at the autopsy; in others, variable symptoms were produced (jaundice, enlargement of the liver, cachexia, hemorrhage, etc.). The diagnosis is only possible if the parasite or its ova are vomited or discharged in the feces.

Distomum hepaticum has a flat, elongated, oval shape, and is 2.5 to 4 cm. long (Fig. 55). The ova are easily recognizable (Fig. 57). *Distomum lanceolatum* is smaller and more slender (Fig. 58), and its ova have a different shape (Fig. 59). These parasites probably enter the intestines in the form of cercariæ in drinking-water or imperfectly cleaned vegetables.

6. *Dropsy of the Gall-Bladder. Hydrops Cystidis Felleæ.*

1. This term is applied to distention of the gall-bladder with a serous or mucin-containing fluid. The disease is the result of occlusion of the neck of the gall-bladder or the cystic duct by gall-stones, adhesion of the mucous membrane, or cancer. The bile retained in the gall-bladder is gradually absorbed and is replaced by a mucin-containing fluid secreted by the mucous membrane of the gall-bladder or a serous fluid from the blood-vessels. The amount of fluid may be remarkably large (sixty to eighty pounds in Erdmann's case). The gall-bladder, filled with synovia-like fluid, is transparent, its walls are not infrequently calcified in places, the mucous membrane is smooth and shiny like a serous membrane, the mucous glands are atrophic or have disappeared; the muscular coat is also atrophied. The organ is sometimes adherent to adjacent organs.

Frerichs found the following substances in the contents of the gall-bladder:

Water.....	98.27
Organic matters (mucus, etc.).....	1.6
Alkalies.....	0.06
Earthy matters.....	0.07
	<hr/> 100.00

2. The chief symptom is the demonstration of a smooth, tense, usually fluctuating tumor in the region of the gall-bladder. The tumor is generally as large as the fist, but occasionally attains the size of the head, and then extends to the pelvis. If the gall-bladder is very tense, fluctuation may be absent. In one case, circular fibres produced a deep constriction at the middle of the tumor, so that it simulated the shape of the kidney. The tumor often presents considerable lateral motion, and accompanies the respiratory movements of the liver. In one of my cases, the transverse colon was permanently situated between the anterior tip of the tumor and the lower border of the liver, so that it was separated from the liver by a tympanitic note on percussion. If the abdominal walls are very thin and the tumor sufficiently large, prominence and respiratory displacemen

may become visible to the eye. Subjective symptoms are often absent, sometimes there is a slight feeling of pressure or tenderness in the region of the gall-bladder.

3. The diagnosis is usually easy, although the disease has been mistaken for ascites. In empyema of the gall-bladder, there is usually fever and cachexia; in cancer of the organ, we find a harder, usually nodular tumor; in distention with gall-stones, there is a feeling of stony resistance and palpable or audible friction of the calculi against one another; distention with bile is usually a more acute condition, and jaundice is generally present; in cancer of the lower surface of the liver, the palpable prominences are hard and nodular, and have a broad base; fever and hectic symptoms are present in abscess of the liver.

4. As a rule, the prognosis is good. Rupture of the gall-bladder rarely occurs from excessive distention, more frequently from injury.

5. Treatment is hardly necessary. Puncture may be performed if the absence of respiratory movements of the tumor or the immobility of the abdominal walls over it, indicates adhesion of the tumor to the abdomen. In the other event, adhesion to the abdominal walls should first be secured as in operations for hepatic abscess.

7. *Accumulation of Pus in the Gall-Bladder. Empyema Cystidis Felleæ.*

The accumulation of pus in the gall-bladder is generally the effect of gall-stones, more rarely of occlusion of the ductus choledochus. If the cystic duct is also obstructed, the pus may distend the gall-bladder into a tumor as large as a man's head.

The physical signs are the same as those of dropsy of the gall-bladder, but severe pain, chill, fever, and emaciation are also present in the former affection. The prognosis is grave. If adhesions to the abdominal walls have formed, the treatment consists of cholecystotomy and removal of the contents of the tumor; in other cases, adhesion to the abdominal walls should first be secured.

8. *New Growths in the Biliary Passages.*

Fibroma and myxoma have been observed in these organs, but a diagnosis is impossible during life.

Cancer alone possesses clinical interest. The tumor may be primary or secondary, the latter either spreading from adjacent organs or developing as metastases.

Primary cancer of the biliary passages is rare, but is more frequent in the gall-bladder than in the ducts. In the former region, it forms a nodular tumor which may attain the size of a child's head. The interior of the gall-bladder contains bile or ichorous masses, and often gall-stones. The other symptoms are: Pain over the tumor, jaundice, vomiting, hæatemesis, diarrhoea, enterorrhagia, and marasmus. Perforation sometimes takes place into the abdominal cavity, duodenum, or colon. The disease generally develops after the age of forty years.

Primary cancer of the bile-ducts causes occlusion, the causes of which cannot be discovered during life.

B. DISEASES OF THE PARENCHYMA OF THE LIVER.

1. *Hyperæmia of the Liver.*

I. ETIOLOGY.—Hyperæmia of the liver may be the result of impeded flow of blood from the organ or of an increased supply of blood. The former is called mechanical or stasis (passive) hyperæmia, the latter congestive (active) hyperæmia.

The causes of passive congestion are: Diseases of the circulatory and respiratory apparatus, diseases in the mediastinum and peritoneal cavity which compress the inferior vena cava, and diseases of the hepatic veins themselves.

Congestion of the liver is often found in uncompensated valvular diseases of the heart. The stasis readily extends to the hepatic veins, because these enter the vena cava in the immediate vicinity of the heart, and also because the blood pressure in the hepatic veins is normally low. Hepatic congestion is especially frequent in mitral and tricuspid lesions, rarer in aortic lesions. Congestion of the liver is also an inevitable result of diseases of the heart muscle and pericardium (myocarditis, fatty heart, pericarditis, synchia pericardii) when these give rise to stasis in the right side of the heart; it is also the result of insufficiency of the heart's action in old age and marantic conditions. Among respiratory diseases, hepatic congestion is especially frequent in pulmonary emphysema, chronic bronchitis, asthma, interstitial pneumonia, pleurisy, and obliteration of the pleura. These diseases entail increased work on the right heart; when the power of this organ fails, stasis is the result. Deformities of the spine and thorax not infrequently produce hepatic congestion by displacing the lungs and heart, and interfering with the function of the latter organ. It is an almost constant effect of asphyxia in the new-born. Aortic aneurism, mediastinal tumors, and cancer of the retro-peritoneal glands give rise to the disease when they compress the inferior vena cava. In very rare cases, the hepatic vein itself undergoes compression. Frerichs observed the formation of valves in the hepatic veins. Stenosis of the hepatic veins is an occasional result of periphlebitis or of thickening and retraction (as the result of chronic perihepatitis) near the entrance of the hepatic veins into the inferior vena cava. Local congestion of the liver may develop in tight-laced fissures, if the circulation below the latter is impeded.

Active congestion of the liver is sometimes physiological, for example, during the period of digestion. This physiological process becomes morbid and permanent, if the ingestion of food is constantly excessive, and sufficient bodily exercise, which aids the circulation in the portal vein, is not taken.

In some cases hepatic congestion is the result of the introduction of irritating substances in the food (condiments, alcoholics), which are conveyed into the portal circulation.

The affection is more frequent in the tropics than in temperate zones. It is not uncommon as the result of infectious diseases, for example, malaria, typhoid and typhus fevers, relapsing fever, pneumonia, erysipelas, scurvy, dysentery, etc.

In some cases it develops (locally or generally) as the result of injury. In some of these cases the congestion passes into inflammation and abscess.

Partial congestion is observed not infrequently around foci of disease in the hepatic parenchyma (abscess, tumors, parasites).

Finally, we must mention vicarious congestion which seems to be produced in a reflex manner through the agency of the vaso-motor nerves. Thus, hepatic congestion may take the place of menstruation, or it develops shortly before the menses appear; it is not infrequent at the menopause. It is often associated with uterine and ovarian diseases, and sometimes takes the place of an hemorrhoidal flux.

Hyperemia of the liver is almost exclusively a disease of adult life. The male sex predominates in some forms, the female sex in others.

II. ANATOMICAL CHANGES.—In passive congestion of the liver the organ is enlarged, sometimes in all directions, sometimes particularly in thickness. The capsule is very tense, and is either entirely smooth or thickened in places. Its free edge is often blunt and round, its notches abnormally deep. The consistence is increased; on pressure, serous fluid sometimes escapes from the cut surface.

The organ contains an unusual amount of blood, and sometimes has a deep blackish-red or steel-blue color. The central veins of the lobules are particularly dilated, and contrast strongly with the pale periphery of

the acini, particularly if the cells in the latter are fatty or pigmented (nutmeg or cyanotic nutmeg liver).

If passive congestion has lasted for a long time, atrophic processes gradually develop (red atrophy of Virchow). The liver, which is still enlarged, has an uneven and nodular surface, and increased firmness. In a few of these cases Frerichs found that the bile contained albumin.

In the simple cyanotic nutmeg liver the central veins of the lobules and the adjacent intralobular capillaries are unusually wide. The dilatation diminishes towards the periphery of the lobules. The enlargement of the vessels causes compression of the cells; they undergo fatty degeneration, become infiltrated with bile pigment, diminish in size and finally disappear, leaving flakes of pigment behind. Connective tissue proliferation develops around the dilated capillaries.

The atrophic, cyanotic nutmeg liver develops when retraction takes place in the newly formed connective tissue. In exceptional cases, this occurs also in the intralobular connective tissue. Brieger noticed the new-formation of biliary capillaries. The atrophic nutmeg liver is often mistaken for true cirrhosis of the liver: the former does not possess a yeast-like color, the irregularities in it are less marked and distributed more irregularly, and enlargement of the spleen is usually absent.

The mucous membrane of the stomach and intestines is often in a condition of stasis-catarrh, and ecchymoses are observed not infrequently. The hemorrhoidal veins are often dilated. The trabecular tissue of the spleen generally undergoes hyperplasia. The pancreas is often very hyperæmic, and unusually firm as the result of interstitial development of connective tissue. The appearances of passive congestion are presented by the kidneys and pelvic organs.

Active hepatic congestion is either partial or diffuse. The liver is uniformly red, peculiarly succulent, and an unusual amount of blood exudes upon pressure.

III. SYMPTOMS.—Passive congestion of the liver is not distinguished clinically until the organ increases in size.

This enlargement is not often sufficient to produce prominence of the right hypochondrium. The lower border of the liver is occasionally found unusually low, and the lower ribs on the right side are raised upward.

The lower edge of the liver is often felt distinctly on palpation. It is unusually firm and round, and extends lower than normal. It is sometimes felt below the umbilicus, or even as low as the anterior superior spinous process of the ilium. Both fissures at the lower edge of the liver can sometimes be distinctly felt.

Percussion reveals increase of the greater (relative) and lesser (absolute) hepatic dulness, especially inferiorly (absolute hepatic dulness normally extends upwards, in the right nipple line, to the lower border of the sixth rib, inferiorly in the median line midway between the ensiform cartilage and the umbilicus). Palpatory percussion often furnishes more certain results.

Physical examination may prove useless if the abdomen is distended by ascites. Palpation may then furnish positive results, if it is performed vigorously and interruptedly with the tips of the fingers, and the fluid between the abdominal walls and anterior surface of the liver is thus pushed aside. Examination in the knee-elbow position may also be useful. The conditions often become very clear if puncture of the abdomen is performed and the fluid removed.

Jaundice is frequent in passive congestion of the liver. It is observed usually in the conjunctiva, sometimes in the face and on the trunk. If the patient is suffering from cardiac disease the face often has a peculiar greenish color.

The jaundice is the result of stenosis of the finer biliary passages by the dilated vessels and of catarrh of the bile-ducts. Severe jaundice is the result of gastro-duodenal catarrh.

The stasis gives rise to gastro-intestinal catarrh (anorexia, vomiting, eructations, constipation, more rarely diarrhœa). Hæmorrhoidal hæmorrhages also occur from stasis in the hæmorrhoidal veins.

Edema, albuminuria, etc., depend upon the primary affection. But ascites is the direct result of the passive congestion of the liver, inasmuch as the atrophic changes narrow the domain of the portal circulation. If ascites exists alone, or is more marked than edema of the subcutaneous tissue, it is evidence of the fact that atrophy has begun in the passively congested liver. In addition, it will be found that a previous enlargement of the liver begins to diminish.

In many cases the subjective symptoms are extremely slight (tenderness in the hepatic region, increased in the sitting position and in right lateral decubitus). Many patients also experience pain when they assume left lateral decubitus. There is often shortness of breath, sometimes severe pains, which may shoot into the right shoulder and arm.

The size of the liver often undergoes rapid changes, sometimes even within twenty-four to forty-eight hours.

In active, diffuse hepatic congestion the local symptoms are similar to those described above; in local congestion they hardly form an object of clinical observation.

IV. DIAGNOSIS.—The diagnosis is easy if we can determine the three following factors: (*a*) The demonstration of the causes of hepatic congestion; (*b*) The presence of hepatic enlargement; (*c*) Rapid changes in the size of the organ.

V. PROGNOSIS.—Hepatic congestion *per se* rarely gives rise to any danger, but the primary disease may render the prognosis unfavorable.

VI. TREATMENT.—The treatment must be chiefly causal. When the disease is the result of insufficiency of the heart's action, digitalis should be administered; in addition, nourishing diet. Four to six leeches may be applied to the anus to relieve the portal circulation. Mild laxatives may also be given for the same purpose (rhubarb, senna, colocynth, aloes). If the patient is not too weak, he may be sent to Kissingen, Homburg, Marienbad, Carlsbad, Tarasp. In marked ascites, as the result of the atrophic nutmeg liver, puncture of the abdomen may become necessary. Diuretics may also be employed, but diaphoretics are contraindicated. Faradization of the abdominal walls has been successful in some cases.

In active hepatic congestion following an injury, the region of the liver should be covered with ice-compresses, leeches applied to the anus, leeches and cups over the liver, and mild laxatives administered internally.

High livers should be restricted as to the amount of food, should avoid spiced articles and alcoholics, exercise a good deal in the open air, and drink the above-mentioned mineral waters. Grape cures and whey cures are also indicated.

If the hepatic congestion is the result of disturbances of menstruation, a few leeches may be applied to the upper inner surface of the thighs or the portio vaginalis, and a warm mustard foot-bath taken at night.

2. *Perihepatitis*.

I. ETIOLOGY.—Perihepatitis is rarely primary, and in almost all such cases is the result of injury. This includes the pressure of the corset.

The affection is more often secondary to hepatic diseases (congestion,

cirrhosis, abscess, cancer, echinococcus). In other cases it forms a part of general peritonitis. Diseases of the stomach, duodenum, colon, and even the kidneys may also give rise to hepatitis, the inflammatory process extending along the ligaments of the liver to its hilus. Perihepatitis may also be associated with right pleurisy. Syphilis sometimes acts as a cause.

II. ANATOMICAL CHANGES.—Perihepatitis may be acute or chronic, circumscribed or diffuse.

In chronic perihepatitis we find opacity and thickening of the capsule, frequently fibrous adhesions between the liver and adjacent organs. The capsule is sometimes so thick as to form a tendon-like, almost cartilaginous covering of the organ. In some cases retraction of the capsule causes slight depressions upon the surface, in others retraction at the hilus causes stenosis or occlusion of the portal vein or excretory ducts, or similar processes near the suspensory ligament produce stenosis of the inferior vena cava and hepatic veins. The shape of the liver is sometimes changed; it becomes smaller and more spherical.

At the beginning of the chronic inflammation, the lymphatics of the capsule are often very widely dilated (perilymphangoitis chronica).

Acute perihepatitis presents the same appearances as acute peritonitis. The capsule is sometimes lifted up from the parenchyma by a circumscribed abscess. Small abscesses have also been observed in the fibrous bands which extend inward from the hilus.

III. SYMPTOMS AND DIAGNOSIS.—In many cases chronic perihepatitis is attended with hardly any symptoms. In others, the disease is recognized by the absence of respiratory movements of the lower border of the liver, on account of adhesions to adjacent organs. Peritonitic friction murmurs are sometimes heard and felt, either with every inspiration or only when the abdominal walls are purposely moved over the surface of the liver. In some cases, finally, the disease gives rise to the symptoms of occlusion of the portal vein, incurable jaundice, congestion or cirrhosis of the liver.

Hambursin has called attention to the fact that, as the result of perihepatic adhesions between the liver and diaphragm, pericardial adhesions may form over the corresponding parts of the upper surface of the diaphragm, and may gradually prove fatal with the signs of insufficiency of the heart's action.

Acute perihepatitis begins occasionally with a chill and fever. The patients complain of pain and tenderness in the hepatic region, and this may give rise to dyspnoea by interference with the respiratory movements. Slight jaundice sometimes develops, also vomiting, anorexia, indigestion. Friction murmurs are less frequent than in chronic perihepatitis.

IV. PROGNOSIS.—In acute perihepatitis, the prognosis depends upon the primary disease; in the chronic form, the prognosis is usually good.

V. TREATMENT.—In the acute form, the patient should keep to bed, a warm poultice applied over the liver, and morphine injected subcutaneously if the pains are very severe. The following measures have also been recommended: cups and leeches over the liver, blisters, tincture of iodine, ice-bags, calomel internally, etc.

Chronic perihepatitis, as a rule, does not require special treatment.

3. *Suppurative Hepatitis.*

(*Abscess of the Liver.*)

I. ETIOLOGY.—This disease is rare in our climate, but much more frequent in the tropics. It is more frequent in men than in women

(30:1), and occurs chiefly in adult life. During childhood it occurs principally in the new-born (from inflammation of the umbilical vein).

Suppurative hepatitis is rarely primary, and then is always the result of injury. The disease is almost always secondary, usually metastatic in character.

It often follows inflammations in the domain of the portal vein (operations on the rectum, diseases of the uterus and ovaries, gastro-intestinal ulcerations, purulent inflammation of the trunk of the portal vein itself). This can only be explained by the conveyance of inflammation-producers from the primary focus through the portal circulation into the liver. This occurs much more rarely through the medium of the hepatic artery, for example, in acute septic endocarditis, gangrene of the lungs, and putrid bronchitis.

In very rare cases the inflammation-producers reach the liver through the hepatic veins, the substances in question passing through the inferior vena cava into the hepatic veins by the action of gravity. This is favored by unusually low pressure in the inferior vena cava. Hepatic abscesses may develop in the manner mentioned if the primary site of inflammation is situated at the periphery of the body (panaritium, venesection wounds, injuries to the bones). The latter are especially dangerous, because the veins in the spongy substance are incapable of collapse, and therefore present a special tendency to the formation of thrombi. Hence, abscesses of the liver occur with relative frequency after injuries to the skull.

In peripheral inflammations, the inflammation-producers may also reach the liver through the vascular system of the lungs. The inflammatory products pass from the periphery to the right heart and then to the lungs, where they give rise to secondary changes. From the latter particles are dislodged, which pass to the left heart, and then through the hepatic artery to the liver. This mode of genesis is only probable if pulmonary and hepatic abscesses are both present. If the former are absent, it is hardly plausible that the inflammation-producers have passed through the pulmonary capillaries and lodged in the hepatic capillaries. In rare cases, perhaps, these elements may pass from the right to the left side of the heart through direct communications between small pulmonary arteries and veins.

Virchow showed that in some cases of peripheral suppuration marantic thrombi form in the vesical, prostatic or uterine plexuses, that these assume malignant properties, and that dislodged particles pass into the liver and produce secondary inflammations.

Suppurative hepatitis may also take its origin in the biliary passages. Thus, gall-stones or ascarides which are incarcerated in the bile-ducts may produce inflammation and suppuration in their walls, and subsequently in the adjacent parenchyma of the liver. According to some writers, this may be the result of simple stasis of bile following catarrh of the bile-ducts. Suppurative hepatitis may also follow ulcerative processes in the walls of the ducts through the agency of the blood-vessels.

Abscess of the liver may also complicate other hepatic diseases (tubercles, echinococci).

In other cases, the disease is propagated from adjacent organs (ulcer and cancer of the stomach, which extend to the liver after the formation of adhesions.)

Finally, a series of cases remains in which the mode of origin cannot be discovered.

Europeans are especially apt to be attacked in the tropics. This has been attributed to the fact that the European in the tropics continues to partake largely of meat and stimulants. Sachs thought that alcohol directly produces, in the tropics, an acute hepatitis. At all events, the influence of climate cannot be denied. Telluric influences are also demonstrable, since certain regions in the tropics are notorious for the frequency of abscess in the liver. Perhaps malaria exercises a certain etiological influence.

II. ANATOMICAL CHANGES.—An abscess of the liver may be situated in any part of the organ, and is either single or multiple. It is more frequent in the right lobe than in the left. As many as forty abscesses have been observed in some cases. On the average, the size of the abscess varies from that of a hazelnut to that of a walnut. In not a few cases it attained the size of a child's head (in Toman's case it contained eighteen pounds of pus). The cavity generally contains laudable pus.

If the abscess has lasted a long time, it sometimes acquires an ammoniacal, more rarely a foetid odor, and the pus may assume a brownish-yellow or chocolate color, from admixture with bile. In rare cases, the pus has a bloody color from rupture of a blood-vessel into the abscess.

The pus usually contains an unusually large number of drops of fat. Its chief constituents are the pus-corpuscles, many of which are in a condition of fatty degeneration; in addition, granular detritus, part of which is composed of bacteria. A few fatty hepatic cells are found occasionally; in one case I observed tablets of cholestearin.

The walls of a recent abscess are irregular and jagged, and occasionally present a creamy, cheesy coating. In older cases the abscess is encapsulated, evidently from proliferation of the interstitial tissue. The capsule is often laminated, and sometimes acquires a cartilaginous consistence. The adjacent blood-vessels are usually obliterated, but a case has been reported in which an aneurism situated on the wall of an abscess gave rise to violent hemorrhage.

The hepatic parenchyma adjacent to the abscess is usually very livid and brittle. In Lepido-Chioli's case the entire organ was waxy. The size of the liver may be nearly doubled.

The biliary passages are not infrequently dilated, and sometimes contain a fibrinous exudation in places. An abscess near the lower surface of the liver may cause general dilatation of the biliary passages by compressing the ductus choledochus and hepaticus. The portal vein may be similarly affected.

In superficial abscesses, the capsule is generally inflamed, and hence adhesions to adjacent organs are formed. In the absence of these, rupture of the abscess would be followed by perforation-peritonitis. If adhesions are present, the perforation may occur into other viscera (stomach, colon, duodenum, rarely into the pelvis of the right kidney, or through the diaphragm into the pleural and pericardial cavities). Adhesions form not infrequently between the pulmonary and parietal pleura, and the abscess may then rupture into the lungs and bronchial tubes. The abscess may also rupture into the portal vein, inferior vena cava, and hepatic veins. It opens occasionally into the large biliary ducts, or the gall-bladder, and then passes into the intestines. Finally, the pus may break through the abdominal walls, either directly or through long fistulæ which may open in the axilla, groin, etc. In rare cases, the pus follows the round ligament to the umbilicus.

If perforation does not occur, the abscess may undergo various changes. These include encapsulation, which may be followed by thickening, caseation, or calcification of the pus. Smaller abscesses are sometimes converted into a retracted cicatrix which contains a calcified nucleus. In rare cases, a cystoid degeneration takes place, the pus assuming a thin, colloid quality.

Recovery after perforation is often rendered difficult from the fact that the walls of the abscess do not approach one another on account of

their rigidity. In very rare cases, gangrenous changes develop when the air enters the abscess.

If the disease is the result of pyæmic or septicæmic infection, abscesses are often found in other organs, particularly the lungs.

The first changes in pyæmic abscesses of the liver consist of distention of the capillaries of the hepatic lobules with bacteria. These proliferate very rapidly, thrombose the vessel, and affect the adjacent cells by pressure and by exerting a chemical influence. The hepatic cells undergo coagulation-necrosis and are finally destroyed. This is followed by the formation of pus. According to some writers, the pus-corpuscles are derived, apart from diapedesis, from the hepatic cells themselves, and from the connective tissue cells of the interstitial tissue.

The original focus is lobular; larger abscesses are produced by the coalescence of smaller ones.

SYMPTOMS.—In not very rare cases, abscess of the liver is found accidentally in the dead-house, the patients never having presented any hepatic symptoms.

In other cases, the abscess produces the symptoms of intermittent fever, usually of the quotidian type (periodical chill, fever, and sweat). A mistake is apt to arise if the spleen is enlarged by previous malaria or pyæmic infection.

Some patients present the symptoms of severe typhoid fever: high fever, clouded sensorium, tympanites, roseola, enlargement of the spleen, delirium, perhaps convulsions or spasms of individual muscles towards the close of life.

A few cases run the course of pulmonary phthisis: gradual emaciation and pallor, night sweats, hectic symptoms.

In another series of cases, the suspicion of hepatic abscess is aroused by suddenly developing symptoms; sudden vomiting of pus, passage of pus in the stools or urine, purulent expectoration, unexpected empyema, or pericarditis, etc.

Cases in which the symptoms are well developed present the following clinical history:

The liver is enlarged, generally in an upward direction (sometimes as high as the second rib). As a rule, this enlargement is not uniform, and percussion of the anterior surface of the thorax reveals convex prominences with the convexity directed upwards. In other cases the enlargement is chiefly or exclusively in a downward direction.

If the liver is much enlarged, the right hypochondrium is more or less prominent. The right lower intercostal spaces are narrowed, the lower ribs raised upwards. In a few cases the epigastric veins are considerably dilated.

An important diagnostic point is the recognition of prominences in the intercostal spaces or beneath the abdominal walls, which undergo respiratory movements so long as no adhesions have formed to the walls of the abdomen.

Palpation reveals the lower border of the liver, which is sometimes remarkably distinct, sometimes is recognized only by the increased feeling of resistance.

There is sometimes circumscribed tenderness in the hepatic region. The entire organ is occasionally tender, but this is more marked in circumscribed regions. Pressure on the liver sometimes causes pain in the right shoulder or gives rise to cough.

One of the most important signs is the demonstration of a fluctuating tumor. This is found to move with respiration so long as no adhesions are present.

If perihepatitis is present, friction murmurs may be heard and felt. There is sometimes unusual tension of the right rectus abdominis, either as a reflex symptom of the pain or as the result of the enlargement of the liver.

Jaundice is absent in the majority of cases, and usually does not appear until the development of the abscess has ceased. It may be the result of pressure on adjacent biliary ducts, catarrh or fibrinous inflammation of the ducts or, in pyæmia, it may be a sign of general infection (hæmatogenous icterus).

Subjective symptoms may be entirely absent. In other cases there is a feeling of fulness and tension in the hepatic region, sometimes intensified into pain, which is situated either superficially or deeply. The pain radiates not infrequently into the right shoulder and arm (through the medium of the phrenic nerve which supplies the liver and also gives off shoulder branches in the distribution of the fourth cervical nerve). In one case the pain in the shoulder was followed by atrophy of the deltoid. According to Annesley, pain in the shoulder occurs only in abscesses of the convexity of the liver. Pain in the left shoulder has been observed in abscesses of the left lobe.

Insomnia and anorexia are not infrequent; mental depression is sometimes observed.

Many patients complain of dyspnœa, the result of interference with the movements of the diaphragm and compression of the lungs by the enlarged liver.

The disease is sometimes entirely apyrexial, in other cases there is a continuous or remittent, hectic or intermittent fever, which may rise above 41° C. Chills often occur, likewise sweats with their sequelæ upon the skin (miliaria, pityriasis tabescentium, defluvium capillorum). The pulse is usually very frequent, small, and soft.

Considerable emaciation is generally noticeable and, together with the pale or yellowish, waxy complexion, gives the patient a cachectic appearance.

According to older writers, abscess of the liver is accompanied not infrequently by cough, vomiting, and eructations. The bowels are often constipated, though diarrhœa, even of a bloody character, has been observed in a few cases.

The patients sometimes lie upon the right side, with the trunk bent over forwards and the right thigh flexed, evidently in order to relieve the liver from pressure.

If the abscess ruptures externally, the skin becomes red, œdematous, and hot, gradually grows thin and bursts. The perforation is sometimes preceded by the formation of vesicles on the integument. The suppuration may continue for a long time, even for years. The fistula sometimes closes completely and re-opens at a later period.

When the abscess ruptures into the stomach, the sudden vomiting of pus is observed, preceded sometimes for days by ordinary vomiting. In Irvine's case hæmatemesis was produced as the result of rupture of an aneurism of the hepatic artery situated in the wall of the abscess.

Purulent evacuations from the bowels indicate rupture into the intestines, either directly into the colon or through the bile-ducts into the duodenum.

Rupture into the pelvis of the right kidney is manifested by pain in the region of the kidney, and by a purulent sediment in the urine. In Huët's case the urine contained numerous liver cells.

Rupture into the inferior vena cava, portal or hepatic veins, gives rise to the signs of internal hemorrhage or metastatic abscess-formation in other organs. Rupture into the peritoneal cavity is followed by signs of diffuse, rarely of circumscribed, peritonitis.

Rupture into the pericardium is followed by pericarditis which usually proves rapidly fatal.

Rupture into the pleura is followed by the signs of empyema, rupture into the lungs by expectoration of pus. The latter often becomes putrid within the lungs and assumes a brownish-yellow or chocolate color. The sputum is sometimes mucous, but contains a larger or smaller number of balls of pus. In a number of cases, the expectoration of pus has been followed by the expectoration of almost pure bile.

It should not be forgotten that pleurisy and pericarditis are sometimes the result of a direct spread of the inflammation without perforation, but the inflammation is then of a serous character.

The duration of the disease is extremely variable. The symptoms sometimes terminate fatally at the end of a few days; in other cases, they may last many years.

IV. DIAGNOSIS.—Despite the exercise of the greatest care, the recognition of abscess of the liver may be impossible.

The best means of differentiation from intermittent fever is the uselessness of the administration of quinine. It is distinguished from pulmonary phthisis by the absence of changes in the lungs, and of elastic fibres and bacilli in the sputum.

If fluctuating prominences are felt on the surface of the liver, they must be distinguished from echinococci, soft, pseudo-fluctuating sarcomas and cystic distention of the gall-bladder.

In echinococci, we must look for the hyatid thrill, in sarcoma for umbilication of the prominences; in dilatation of the gall-bladder, the tumor is pear-shaped and very tense. In addition, puncture should be made with a very fine trocar.

Fluctuating prominences in the hepatic region may also depend on tuberculosis of the ribs or spine, or abscess of the abdominal walls. In these conditions, there is no respiratory movement of the tumor. In the two former conditions pain is felt along the ribs or spine. To distinguish hepatic abscess from one situated in the abdominal walls, Sachs recommended the introduction of a long Carlsbad needle. In the former disease, the head of the needle will present respiratory movements; in the latter, these are absent.

The disease may be mistaken for pleurisy if the upper border of the liver is unusually high, but in abscess of the liver the dulness is irregular, and is usually higher anteriorly.

If pus is suddenly evacuated, we must attempt to convince ourselves of the absence of purulent inflammation in the organ through which the abscess has opened. On microscopical examination, the pus will usually be found to contain liver cells or bile-pigment.

V. PROGNOSIS.—The mortality in abscess of the liver is about eighty per cent. Spontaneous recovery occurs in exceptional cases. Early operative interference has reduced the mortality to forty per cent.

VI. TREATMENT.—The treatment belongs to surgery, not to internal medicine. As soon as the diagnosis has been made with certainty, the pus should be removed. Concerning the methods of operation, we must refer the reader to the text-books on surgery.

4. *Chronic Interstitial Inflammation of the Liver.*

(*Cirrhosis of the Liver. Chronic Interstitial Hepatitis.*)

I. ETIOLOGY.—The disease is generally the result of the excessive ingestion of alcohol (hence the term whiskey liver). The alcohol is probably conveyed to the liver through the portal vein, and there sets up an inflammatory process in the interstitial tissue.

Some authors believe that, as the result of chronic gastro-enteritis produced by the alcohol, abnormal products of decomposition of the food are conveyed to the liver, where they give rise to inflammatory changes. It has also been assumed that the inflammation may spread from the gastro-intestinal tract to the liver along the sheaths of the portal vein.

The more undiluted the alcohol the greater is the danger of cirrhosis. The disease rarely results from drinking beer or wine.

The disease is more frequent in males, particularly from the ages of thirty to sixty years.

A number of cases have been reported in which cirrhosis of the liver was observed in children who had been in the habit of drinking brandy from an early age. Wilkes reported a case in a girl *æt.* 8 years.

Budd believed that other irritating substances in the food may act in the same way as alcohol in producing the disease. He attributes its frequency in India to the habitual use of strong spices (curry, etc.).

Cirrhosis of the liver sometimes appears to be produced by infectious diseases. Thus, it is relatively frequent after intermittent fever, and, according to Frerichs, it is sometimes the result of constitutional syphilis. Proliferation of the interstitial connective tissue of the liver, which is usually recognized only with the aid of the microscope, is observed almost constantly in the course of miliary tuberculosis. Whether the disease may be the result of typhoid fever, cholera, or gout, is still a matter of dispute.

In some cases, it seems to start from the biliary passages, and develops after occlusion of the bile-ducts by gall-stones or tumors, but the cases hitherto reported are not entirely free from doubt.

Botkin and Salowieff have attributed cirrhosis of the liver to occlusion of the portal vein. In some cases, it is the result of the extension of chronic perihepatitis.

Bamberger noticed the development of the disease after menstrual disturbances, Murchison after hemorrhoidal hemorrhages.

Weber and Virchow reported two cases of foetal cirrhosis, but these were probably the result of syphilitic changes.

Typical cirrhosis has been produced experimentally in animals by the prolonged administration of phosphorus (toxic cirrhosis).

In not a few cases, the etiology remains unknown. In a number of cases, I have noticed that the disease developed apparently spontaneously beyond the age of forty-five years, terminated unfavorably quite rapidly, and, at the autopsy, was found to be associated with proliferation of the interstitial connective tissue of the kidneys, and with signs of endarteritis obliterans and arteriosclerosis in the vessels of many organs. I am inclined, therefore, to assume a senile (arteriosclerotic) cirrhosis of the liver, which appears to depend on senile changes in the vessels.

II. ANATOMICAL CHANGES.—The inflammatory process in this disease begins in the interlobular connective tissue, in the majority of cases

in the immediate vicinity of the medium-sized and smaller branches of the portal vein.

The connective tissue increases in amount, encircles single or, more frequently, several lobules, and causes fatty degeneration and disappearance of the liver cells by the increasing pressure. On section, the abnormally broad bands of connective tissue are readily recognized, and between them the remains of the parenchyma project above the surface, so that the latter assumes a nodular and granular appearance. This appearance is also presented by fatty liver, atrophic nutmeg liver, and pylephlebitis adhesiva.

There are two stages of cirrhosis of the liver. In the first, the organ is enlarged; in the second, it is small and retracted. The latter is the result of retraction of the newly-formed connective tissue. But the processes of hyperplasia and retraction are associated with one another to such a degree that we may long remain in doubt whether the disease should be considered to be in the first or second stage. In some cases, indeed, the organ never grows smaller (hypertrophic cirrhosis). In the so-called second stage the liver presents the following changes: It is diminished in size, and not infrequently is only one-third the normal dimensions. The weight may be diminished from 50-65 ounces to 35 ounces. The retraction affects the left lobe to the most marked extent. The liver is not infrequently adherent to adjacent organs (stomach, colon, duodenum, diaphragm, etc.). Its surface is uneven and nodular, the individual prominences varying from the size of a pea to that of a hazelnut. Some prominences may be almost entirely separated from the rest of the organ. The capsule is thickened in places, especially where the prominences coalesce. Upon section, the capsule is often found to send firm bands of connective tissue into the interior of the liver. The gall-bladder usually contains but little bile, and this is generally very light, almost straw-yellow in color.

The liver is increased in consistence, and not infrequently creaks when cut with the knife. It has a bright reddish-yellow color, partly from fatty degeneration, partly from the marked amount of pigment in the hepatic cells. In rare cases portions of the organ have a blackish-gray or greenish-black color.

The lower border of the liver is blunt and rolled upwards, more rarely downwards.

Injection of the liver with colored substances will produce different effects, according as the injection-canula is inserted into the portal vein or hepatic artery. In the former event the fluid soon ceases to flow, whence it may be inferred that the fibrous retraction has led to occlusion of branches of the portal vein within the parenchyma; while, in the latter event, the injection-fluid readily enters the branches of the hepatic artery. The biliary passages may be readily injected, in almost all cases, through the hepatic duct.

The liver almost always presents the various stages of development of the chronic inflammatory process. In parts which have been recently affected, the interlobular connective tissue is broad and infiltrated with numerous round cells, particularly around the branches of the portal vein. These are probably derived partly from the blood, partly from division of pre-existing connective-tissue cells. It is even believed by some that the liver cells may be converted into connective-tissue cells.

At a later period this cellular infiltration is converted into connective tissue.

In the beginning, the connective-tissue hyperplasia usually encircles several lobules, but later it extends into the lobules themselves. Round cells continue to emigrate from the capillaries between the liver cells, compress the latter, and cause them to disappear, are finally converted into connective tissue, and thus replace the destroyed parenchyma.

The liver cells are thus compressed from the periphery and from the interior of the lobules. In addition, some of the branches of the portal vein are occluded by the interlobular proliferation of connective tissue, and hence the hepatic cells are partly deprived of their supply of blood. The most marked changes are observed at the periphery of the lobules, where the cells are filled with drops of fat and bile pigment, in yellowish or brownish granules or in fine needles. The individual cell gradually disappears, and leaves only a little bile pigment. In this way many lobules may be entirely destroyed.

The vascular system of the liver, particularly that of the portal vein, also undergoes changes. Numerous branches are narrowed and finally occluded as the result of endophlebitis obliterans. The same process may take place in the intralobular capillaries. In the distribution of the hepatic artery, on the other hand, numerous capillaries may develop, and may form communications with the remains of the branches of the portal vein.

The biliary canals also undergo proliferation. The interlobular connective tissue is traversed by numerous biliary canals; the epithelium of these new-formed vessels is said to develop from rows of hepatic cells.

The occlusion of the branches of the portal vein explains the symptoms of stasis (splenic enlargement, ascites, gastro-intestinal catarrh, etc.), the non-affectation of the biliary canals explains the frequent absence of jaundice.

In rare cases the hepatic veins, and even the inferior vena cava may be narrowed, inasmuch as the inflammatory proliferation extends to the walls of these vessels.

Among the complications of this disease we may mention waxy degeneration, in rare cases, cancer, abscess, or echinococcus. In two cases Bamberger observed acute atrophy of the liver in places in a cirrhotic liver.

According to Ackermann, the process begins in the liver cells, and is followed at a later period by increase of the interstitial connective tissue. Charcot and Gombault distinguished three forms of cirrhosis :

- a. Cirrhosis starting from the veins.
- b. Cirrhosis starting from the biliary passages.
- c. Monocellular cirrhosis.

That form which starts from the veins corresponds to that described above. In that variety which starts from the biliary passage the new-formed connective tissue forms scattered foci, and from the beginning involves each lobule separately. It is said that this form of the disease may be produced experimentally in animals by ligature of the bile-ducts, occurs in man as the result of occlusion of the ducts, and that in some cases it occurs as an independent disease. Olivier and Hayem called this disease hypertrophic cirrhosis, because the organ is enlarged and presents no tendency to subsequent atrophy. The liver and spleen undergo considerable increase in size; icterus is a prominent symptom, and the disease is very protracted.

Monocellular cirrhosis begins with intralobular and extralobular proliferation of connective tissue, which surrounds single hepatic cells. This form includes many cases of syphilitic changes in the liver.

English and German authors have protested against the strict acceptance of these views. To a certain extent they are founded on truth, but there are so many transitions between the different forms that they cannot be regarded as distinctly separate.

In fatty hypertrophic cirrhosis, the accumulation of fat in the hepatic cells is so extensive that the organ is increased in size.

Simple induration of the liver is the term applied to a chronic process of proliferation of the connective tissue, in which large foci of hepatic tissue are entirely destroyed and replaced by connective tissue. In this condition many branches of the portal vein undergo obliteration. The disease cannot be distinguished during life from cirrhosis of the liver.

The initial changes in cirrhosis are not often observed on autopsy. The liver is enlarged one to three fold, is unusually firm, and on section we notice numerous broad, gray bands of connective tissue, which traverse the parenchyma.

The abdominal cavity usually contains a considerable quantity of a serous, more rarely of a hemorrhagic transudation. Extravasations of blood not infrequently are found here and there upon the peritoneum. In places the peritoneum is

thickened, opaque, and even œdematous. The portal vein sometimes contains adherent thrombi. The spleen is usually enlarged, firm, and its capsule thickened in places; on section the trabecular tissue is found to be abnormally developed. The gastro-intestinal mucous membrane presents evidences of chronic catarrh, not infrequently extravasations of blood. In several cases Fraentzel observed phlegmonous gastritis. The kidneys are often congested, not infrequently cirrhotic or in a condition of parenchymatous inflammation. Fatty degeneration, the formation of callosities, and endocarditic changes are found in the heart. Hydrothorax and hydropericardium are found not very infrequently. The lungs present evidences of bronchitis, emphysema, œdema, and inflammation.

III. SYMPTOMS.—The first symptoms are usually masked by those of obstinate gastro-intestinal catarrh (anorexia, eructations, coated tongue, feeling of pressure in the stomach, etc.).

An affection of the liver is not recognized until the organ has undergone changes in size.

The liver is at first enlarged; later it grows smaller. The increase in size is usually most marked in the downward direction, so that the lower border of the organ can be seen and felt far below the umbilicus. Atrophy of the liver is especially marked in the left lobe, whose dulness is absent or very much diminished in extent, and is replaced by the tympanitic note of the stomach.

If perihepatitis is present, peritonitic friction murmurs are sometimes felt and heard. The nodules on the surfaces are sometimes felt on palpation in lean individuals, but we should avoid mistaking the nodules for the panniculus adiposus. It should be remembered that the former move with respiration.

The development of ascites, without œdema of the lower limbs, is a very important symptom in cirrhosis, although it only indicates obstruction to the circulation in the portal vein. We must therefore exclude diseases of the portal vein itself (pylephlebitis, compression by tumors, exudations, fibrous cicatrices) or of the peritoneum (cancer, tuberculosis, serous peritonitis). It is said that ascites is usually absent in hypertrophic cirrhosis.

When the patient, at the first examination, presents marked ascites, the diagnosis is rendered difficult on account of our inability to determine the size of the liver. This can sometimes be done by placing the patient on the left side or in the knee-elbow position, and thus displacing the fluid which is situated in front of the liver. After puncture of the abdomen and discharge of the fluid, the outlines of the organ usually become extremely distinct, but at the end of a few hours or days the fluid has often accumulated to such an extent that the organ is again submerged.

The amount of fluid is often astonishing; its color varies from an amber-yellow to a bile-stained, sanguinolent, or distinctly bloody color. The blood-corpuscles contained in it may be entirely dissolved. The specific gravity varies from 1010 to 1015; the fluid contains 2 to 3 per cent of solid constituents.

Enlargement of the spleen is the third one of the cardinal symptoms in cirrhosis of the liver. It may be absent if the capsule has been previously thickened and calcified. The dimensions of the organ may be increased four to six fold.

The splenic enlargement is chiefly the result of stasis in the portal circulation. In some cases, however, the connective-tissue proliferation in the spleen seems to result from the same causes as that in the liver. In a few cases, abscess and infarctions have been found in the organ.

Jaundice may be absent during the entire course of the disease, because the biliary canals are not affected in such a manner as to encroach

materially upon their lumen. Early and marked jaundice will occur only in those rare cases in which the hepatic affection is the result of stenosis of the bile-ducts. In the majority of cases, the jaundice is confined to the sclera.

Icterus should be distinguished from the grayish-yellow, sallow complexion observed in most of the patients. The integument is generally lean, thin, and dry. Marked emaciation generally develops at an early period. (Edema of the skin may be entirely absent, but at a late period in the disease slight œdema is usually observed in the lower limbs as the result of marasmus, pressure of the ascites on the inferior vena cava, or implication of this vein (as the vessel passes across the posterior border of the liver) in the process of retraction.

The abdominal walls often present abnormal dilatation and sinuosity of the cutaneous veins. The inferior epigastric veins ascend from about the middle of Poupart's ligament, and unite at the level of the umbilicus with the superior epigastric veins; the latter unite near the thorax with the mammary veins. The dilatation is the result of compression of the inferior vena cava, so that a portion of the blood from the lower limbs reaches the heart by circuitous routes.

Unusual vascular channels are sometimes opened as the result of occlusion of branches of the portal vein (*caput Medusæ s. cirrhophalos*). Under such conditions, a portion of the blood in the portal vein flows through the open umbilical vein, which is generally unused, passes externally at the middle of the umbilicus, and there communicates with the adjacent epigastric veins. The umbilical vein and its external ramifications undergo varicose dilatations and sometimes give rise to a purring thrill and an almost continuous venous hum.

Various other collateral communications may be formed between the portal vein and the right heart, for example, *a.* Left coronary vein of the stomach—inferior œsophageal veins—*vena azygos*. The œsophageal veins may also undergo varicose dilatation, and thus be the source of dangerous hemorrhage. *b.* Veins of the capsule of the liver or gall-bladder—diaphragmatic veins. *c.* New-formed veins in the fibrous adhesions—diaphragmatic veins. *d.* Sappey's accessory veins, which run from the surface of the liver along the round ligament to the abdominal walls—epigastric, mammary, and superficial abdominal veins. *e.* Mesenteric veins—veins of the abdominal walls. *f.* Mesenteric veins—spermatic veins. *g.* Internal hemorrhoidal vein—hypogastric vein.

Abnormal communications may also develop. Virchow reported a case in which an anastomosing varix developed between the splenic vein and *vena azygos*.

The patients usually suffer from anorexia, and often from increased thirst. The tongue has a grayish-white, or brownish-yellow coating. Singultus and vomiting are observed not infrequently. Very marked disturbances of respiration are often produced by meteorism associated with ascites. As a rule, there is constipation, more rarely diarrhœa. Hemorrhoids are found only in rare cases.

If the symptoms of stasis increase, hemorrhages are sometimes produced (*hæmatemesis*, hemorrhoidal hemorrhages).

Heiller reported a case in which *hæmatemesis* was the first symptom of the disease. In Rollet's case, this symptom occurred at almost regular intervals of four to five weeks' duration. Schilling reported a case in which fatal *hæmatemesis* was the first and only symptom of cirrhosis of the liver. Ascites and enlargement of the spleen are apt to diminish after hemorrhages.

The urine is almost always diminished in amount; its specific gravity is increased, its reaction very acid, and on cooling it very often deposits

a red sediment of urates. If jaundice is present, biliary coloring matter and biliary acids are found in the urine. Langenbeck observed hemorrhage from the bladder in two cases. A small amount of albumin in the urine must be attributed to cachexia and stasis produced by ascites, a large amount of albumin to nephritis. In the latter event, the presence of casts is important. In cases of jaundice, a few hyaline casts may be present, despite the absence of nephritis.

According to Andiguer, the amount of urea in the urine is diminished; Debove claims that, when the urea is diminished in the urine, it is increased in the blood. The chlorides in the urine have also been found diminished, the ammonia increased.

The urine sometimes contains sugar. French writers claim that the abundant ingestion of sugar in cirrhosis of the liver gives rise to glycosuria, and explain this on the theory that a portion of the sugar is not converted into glycogen within the liver, on account of the occlusion of branches of the portal vein, and destruction of liver-cells.

Respiration may be seriously interfered with as the result of extensive ascites; bronchitis and pneumonia develop quite frequently. Gée and Galliard observed hæmoptysis; the latter writer also observed hæmatoma of the pleura.

The heart is not infrequently displaced as the result of ascites. Signs of dilatation of the heart, particularly of the right side, cardiac insufficiency, and valvular lesions may be noticed. The peripheral arteries are often in a condition of arteriosclerosis.

The following ocular symptoms have also been observed: retinal hemorrhages, retinitis pigmentosa, xanthopsia.

The average duration of the disease is one to three years, though it occasionally lasts five years, or even more. The disease is usually apyrexial, but Riva described intermittent attacks of fever, which he attributes to disturbances in the function of the liver. Fever often develops towards the close of life.

Death occurs not infrequently from increasing marasmus, sometimes preceded by the signs of dissolution of the blood. It is sometimes hastened by violent diarrhœa or gastro-intestinal hemorrhage. Towards the close of life the lower limbs usually become œdematous. In other cases the ascites becomes excessive, and proves fatal by interfering with circulation and respiration.

Intercurrent bronchitis, pneumonia, or heart failure is sometimes the immediate cause of death.

In rare cases death occurs with symptoms of cholæmia. The patients become comatose, restless, delirious, are attacked by general convulsions or twitchings, the temperature often rises, and a typhoid condition develops which terminates in death.

When intense jaundice is present, cholæmia appears to be due to blood-poisoning with the constituents of the bile. It may also be the result of acholia, the liver ceasing to secrete bile, and injurious excrementitious substances being thus retained in the blood.

In rare cases the scene terminates with the symptoms of acute yellow atrophy of the liver.

IV. DIAGNOSIS.—The diagnosis can only be made with certainty if it is favored by the etiology (alcoholism, etc.), if an enlarged liver gradually grows smaller, and if ascites and splenic enlargement are demonstrable. Demonstrable atrophy generally does not occur until after the lapse of weeks. In one case Stricker observed a diminution of eleven

cm. in the area of hepatic dulness within four weeks. Ascites must often be relieved by puncture of the abdomen, before a diagnosis can be made. The disease is most frequently mistaken for the following affections :

a. Waxy liver. Enlargement of the liver and spleen, and ascites are also present in this affection, but the etiology is different (suppuration, cachexia, etc.); the liver is smooth and very tense; the urine contains a large amount of albumin; there is marked œdema of the limbs, often of the face; jaundice is absent unless the excretory ducts are compressed by degenerated glands.

b. Cancer of the liver. Enlargement of the liver and ascites are often present, but splenic enlargement is rarely observed; in addition, rapidly increasing cachexia, cancer in other organs, advanced age of the patient.

c. Syphilis of the liver. This is distinguished by the history and the presence of syphilitic changes in the skin, mucous membrane, and bones.

d. Pylephlebitis adhæsiva. Symptoms of stasis develop rapidly, and the etiology is different.

e. Congestion of the liver, vide page 191.

f. Chronic peritonitis; usually attended with diffuse, marked tenderness of the abdomen.

V. PROGNOSIS.—This is always unfavorable.

VI. TREATMENT.—If we suspect the beginning of cirrhosis in drunkards, we should warn them against the abuse of alcohol, prescribe easily digested, unirritating food; and a cure in Carlsbad, Kissingen, Homburg, etc. Mercury internally and by inunctions, leeches and cups over the liver, leeches around the anus, blisters, warm and cold compresses over the liver have also been recommended.

If the disease is at its height, the diet should be nutritious, but the patient should not be deprived entirely of alcohol, in order to avoid collapse. The milk-cure is said to have produced improvement, and even recovery in some cases. In the treatment of ascites too much should not be expected from the administration of diuretics, diaphoretics, and drastics. Puncture of the abdomen should not be looked upon as a last resort. The operation should be performed relatively early, and, if necessary, should be repeated. In one case Bamberger repeated the operation eleven times in two and a half months, and thus removed three hundred and fifty pounds of fluid.

5. *Acute Yellow Atrophy of the Liver.*

(*Acute, diffuse parenchymatous hepatitis. Hepatitis cytophthora.*)

I. ETIOLOGY.—The disease is probably the result, in many cases, of infection with bacteria.

The affection is extremely rare. It may be primary or secondary, the former variety occurring as an independent disease, the latter being secondary to another hepatic affection, or to diseases of the entire organism. The primary form is more frequent in women than in men (1.6 : 1). As a rule, it affects individuals from the twenty-fifth to the fortieth years, but occasionally beyond the age of sixty years or in childhood (earliest case in an infant æt. four days). No cases have been observed between the ages of five and nine years.

It is especially frequent during pregnancy (fifth to eighth months), rarer after parturition. It is hardly ever observed during the first three months of pregnancy.

A sort of epidemic spread has sometimes been noticed. Arnould observed it in ten soldiers who lived in the same wing of the barracks.

Mental emotions and sexual excesses have been regarded as the exciting cause in certain cases. In the large majority of cases no exciting cause is ascertainable.

Some authors believe that phosphorus poisoning may give rise to acute yellow atrophy of the liver, but it is at least questionable whether the changes in these two affections are identical with one another.

Secondary yellow atrophy may be associated with cirrhosis and fatty liver, but it only affects parts of the organ. It has also been observed after certain infectious diseases (typhoid and relapsing fever, pyæmia, etc.). In one case it followed pharyngeal diphtheria, and the hepatic affection was, perhaps, secondary to diphtheria of the stomach. Syphilis and mercurialism are also said to favor the development of the disease.

II. ANATOMICAL CHANGES.—The liver is diminished in size, its tissue is flaccid, and it has an ochre, saffron, or rhubarb color.

The organ may be diminished to one-half or two-thirds, in some cases to one-fourth of the normal size; its thickness is especially affected. It has usually fallen backwards upon the spinal column, so that the anterior surface is covered by the intestines. The capsule is wrinkled because its dimensions are comparatively too large.

The liver is peculiarly flaccid and almost fluctuates on being moved to and fro. In some cases it presents an almost uniform ochre yellow color, in others more or less yellowish-brown or reddish-brown parts alternate with one another. The red portions constitute a later stage of the disease. Since the changes are most marked in the left lobe, this portion sometimes has a reddish-brown color, while the right lobe is yellow. The yellow portions project above the cut surface, the red ones are depressed; the former are brittle, the latter tough and leathery.

The gall-bladder is sometimes almost empty. In other cases it has mucoid, faintly yellow, more rarely greenish contents.

In the yellow portions the liver cells are in a condition of more or less marked fatty degeneration and destruction, particularly at the periphery of the lobules. Finally, nothing remains but a detritus of larger and smaller drops of fat. In places we notice bilirubin crystals, occasionally fine needles of tyrosin.

In places in which the disease was beginning, Frerichs found an exudation between the liver-cells. It is also said that the degeneration of the cells is preceded by cloudy swelling.

The vessels contain small amounts of blood. Injection of the hepatic veins is unsuccessful because the injection mass at once extravasates. Frerichs found needles of tyrosin in these veins.

The reddish-brown spots are developed from the yellow ones by the gradual absorption of the fatty detritus. Finally a basement substance remains containing a few cells and fatty granules. The latter are occasionally found within the intra-acinous blood-vessels and lymphatics, and the walls of the latter may also present fatty degeneration.

The biliary canals may present processes of proliferation. Nuclear proliferation has been observed often in the interlobular tissue, particularly near the branches of the portal vein.

Bacteria have been repeatedly found in the diseased parts. If the liver remains in the open air for several days it becomes covered with a whitish coating of leucin and tyrosin.

In the fresh liver Salkowski found 2.51 per cent peptone and 0.36 per cent hemialbumose, while the spleen contained 2.39 per cent peptone, and 0.58 per cent hemialbumose, and the kidneys 1.8 per cent peptone, and 0.2 per cent hemialbumose.

The blood is usually thin, of a tarry, black color, small in quantity, and occasionally contains a large amount of urea and tyrosin.

Extravasations of blood are found in many organs. The periportal, retroperitoneal, and mesenteric glands are sometimes swollen and congested. The lymph-follicles of the intestines are rarely affected.

The spleen is usually large, soft, and flaccid. The gastro-intestinal tract presents signs of catarrh; the ductus choledochus is sometimes occluded by a plug of mucus. The epithelium cells of the renal tubules are in a condition of fatty degeneration, and sometimes contain hæmatoidin crystals, leucin, and tyrosin. The heart muscle is in a condition of fatty degeneration. Dropsy of the pleura and pericardium has been repeatedly observed. Œdema and inflammation have been observed in the lungs and œdematous changes in the brain. Finally, the voluntary muscles may undergo fatty degeneration.

III. SYMPTOMS.—The prodromal stage of the disease consists of the symptoms of gastro-intestinal catarrh (anorexia, nausea, vomiting, malaise, occasionally slight abdominal tenderness). After a while jaundice usually develops, at first in the face and neck, and gradually extends over the rest of the body. This stage lasts from a few days to several weeks.

Then the patients become restless, begin to grow delirious and violent, the sensorium becomes more and more clouded; grinding of the teeth, attacks of trismus, twitchings in the muscles, or general convulsions make their appearance. The somnolence increases to coma, respiration becomes irregular and stertorous, and the patients finally die in complete coma.

In addition to the nervous system, many other organs are also affected.

The liver undergoes a rapid diminution in size. As the anatomical changes begin earliest in the left lobe, dulness first disappears in the epigastrium, and gives place to a tympanitic note. Then the lower border of the liver gradually extends upwards, and, in marked cases, the hepatic dulness is confined to a narrow strip in the right axillary line. The hepatic region may even be depressed.

The atrophy of the liver is sometimes preceded by enlargement of the organ. In rare cases the atrophy is very slight or entirely absent, especially if the liver had been very fatty or exhibited considerable interstitial proliferation of connective tissue.

Pain in the liver was absent in none of my cases. Despite the impairment of consciousness, the patient manifested pain when pressure was exercised upon the liver. Some authors believe that this is the result of general hyperæsthesia of the skin.

Enlargement of the spleen is a very frequent, though not a constant symptom.

It remains absent if the capsule is thickened or calcified. In some cases it is prevented by hemorrhages from the stomach and intestines. The splenic enlargement is the result in part of the disturbances of circulation in the portal vein, in part of the general infection.

Changes in the integument are almost always present. Jaundice is not a necessary symptom in this disease, and its intensity does not always correspond to the severity of the nervous symptoms. A roseolar eruption has been repeatedly observed. Petechiæ and larger extravasations of blood are sometimes present, and are usually associated with hemorrhages in other regions.

Very important changes are found in the urine. It is greatly diminished in amount, and anuria may occur towards the end of life. The reaction is acid; the specific gravity varies from 1.012 to 1.030. The urine has an icteric color, and also contains biliary acids. It usually deposits a flocculent, brownish or green-

ish sediment, and the presence of leucin or tyrosin in it is almost pathognomonic (vide Fig. 60). These substances can usually be extracted if a drop of urine is placed on an object glass, a little acetic acid added, and then allowed to evaporate. The sediment also contains casts (hyaline or fatty) and fatty, often icteric epithelium from the renal tubules. The urea is absent or greatly diminished. Frerichs noticed an increase of kreatin, absence of phosphate of lime, and considerable increase of extractive matters. Schmeisser noticed the absence of soda salts and a large amount of lime. Riess and Schultzen found lactic acid and peptonoid bodies in the urine. Albumin is found not infrequently, but usually in small amounts.

The disease is almost always accompanied by gastro-intestinal symptoms. The patients often vomit coffee-ground-like, bloody masses. Towards the end of life, this symptom is superseded by obstinate singultus. The tongue has a whitish-gray or brownish-yellow coating; the lips and gums are often covered with sordes.

FIG. 60.



Leucin drops and tyrosin needles from the urinary sediment in acute yellow atrophy of the liver. Enlarged 275 times.

The stools are dry and deficient in bile.

Dissolution of the blood is often shown by hemorrhages into the various organs (stomach, intestines, nose, respiratory tract, skin, etc.).

Riess and Schultzen found tyrosin in the blood. Rosenstein noticed increase of the white blood-globules, amœboid movements and even constrictions of the red globules, and a great tendency to the formation of "raspberry" prominences.

The pupils are usually dilated, and their reaction is slow. Xanthopsia and bilateral amaurosis have been observed.

Fever may be absent, and occasionally the temperature is subnormal towards the end. In other cases, there is an elevation of temperature towards the close of life, occasionally even hyperpyretic temperature (above 41° C.).

At first the pulse is often slow; later it becomes frequent, small, and finally barely perceptible.

The disease is almost always acute, and sometimes lasts only a few days. Death occurs, in the majority of cases, within the first two weeks. In a few cases, death ensued as late as the eighth week, in one case in the fourteenth week.

Those writers who regard acute yellow atrophy of the liver as a general disease lay stress on its occasional epidemic development, the implication of other organs, and the disproportion which may exist between the relatively slight local changes and the severe general symptoms. We regard the disease as a local affection of the liver. Its epidemic occurrence may be explained on the ground that the noxious agent affects several persons at the same time. In other affections of the liver, we also find that other organs are implicated (probably from poisoning with biliary acids).

Numerous theories have been advanced with regard to the real cause of the disease, but none possesses any solid foundation.

The anatomical changes in the liver we regard as inflammatory in character, not as a simple degeneration. This view is favored by the fact that the atrophy of the organ is often preceded by enlargement, by the occurrence of nuclear and cellular proliferation in the interlobular tissue, etc.

The jaundice may be explained by the occlusion of the biliary capillaries by the fatty and granular detritus of the liver cells. The severe nervous symptoms are the result of the accumulation in the blood of excrementitious matters which are normally excreted by the liver and kidneys.

IV. DIAGNOSIS.—The diagnosis is usually easy. In grave icterus from other causes, a mistake may arise if the transverse colon, distended with gas, is situated between the anterior surface of the liver and the abdominal walls, and thus artificially diminishes the hepatic dulness. But this condition is usually not permanent; in addition, vigorous percussion will enable us to hear the hepatic dulness. The differential diagnosis from phosphorus poisoning may be difficult; we must rely upon the history and the demonstration of phosphorus in the contents of the stomach, or, after death, in the other organs.

V. PROGNOSIS.—It is doubtful whether recovery from this disease ever takes place.

VI. TREATMENT.—This must be confined to symptomatic measures.

6. *Fatty Liver.*

I. ETIOLOGY.—An unusual amount of fat in the liver may be the result either of an excessive supply in the portal vein or of unusually vigorous metamorphosis of albuminoids in the liver cells, one part of which is converted into urea, the non-nitrogenous part into fat. The former constitutes fatty infiltration, the latter fatty degeneration of the liver.

Fatty infiltration of the liver is found in individuals who eat largely, particularly of starchy and saccharine food and alcoholics, and take but little exercise. Fatty liver is also one of the alcoholic diseases. It is usually associated with other signs of obesity.

In recent times, the views concerning the formation of fat have been materially changed. It has been shown that it may be formed of albuminoids, which are converted into nitrogenous and non-nitrogenous components. It has been rendered doubtful whether the fats are produced by the carbohydrates.

The fatty liver of drunkards is explained on the theory that the alco-

hol diminishes oxidation, so that less fat is oxidized, and an unusually large amount remains in the liver.

Fatty degeneration of the liver results if a poverty of oxygen is produced by general or local causes and the hepatic cells are also involved. Their albuminoid constituents then undergo the changes mentioned above and the fat then remains in the cells because, on account of the deficiency of oxygen, it does not undergo further oxidation.

This is observed in anæmic and cachectic conditions and after extensive hemorrhages, because the blood is poor in red blood-globules, which act as carriers of oxygen.

Fatty liver is especially frequent in phthisis, particularly in females. The accumulation of fat in the liver cells of phthisical individuals is evidently favored by the slight oxidation of fat in this disease.

Fatty liver is also observed in cancer, chronic diarrhoea, rachitis, scrofula, pernicious anæmia, chlorosis, leukæmia, spinal cord diseases attended with bed-sores, bone suppuration, malarial and syphilitic cachexia. This category also includes the fatty liver of pregnant and purperal women, the so-called acute fatty degeneration of the new-born, etc.

Abnormally high temperature of the body is sometimes the cause of fatty degeneration of the liver cells. This change is often preceded by cloudy swelling.

The process of infection itself predisposes to the disease so that it is sometimes found in infectious diseases which run an apyrexial course.

Marked fatty degeneration of the liver is found after poisoning with phosphorus, arsenic, antimony, etc., because these substances interfere with the absorption of oxygen.

The disease also is produced by stasis in the hepatic veins, since this interferes with tissue respiration. In a case of this kind Frerichs found the first accumulation of fat in the immediate vicinity of the central veins.

Finally, it may be produced by local diseases of the liver (cirrhosis, abscess, etc.).

II. ANATOMICAL CHANGES.—Slight grades of fatty liver can only be recognized with the aid of the microscope. The organ may increase to double the normal size, the edges are usually thickened and blunted. In the fresh condition the liver is soft and compressible; it has a pale or sallow yellow color. The capsule is smooth and tense, and its vessels may be dilated. The gall-bladder sometimes contains only a small quantity of a slightly bile-stained fluid, which may be chiefly mucoid.

The organ is pale on section. The markings are often wiped out, in other cases the centre of the lobules is deeply stained with bile. If the disease is the result of stasis, the centre of the lobules has a blood-red or brownish-red color. The fatty changes are occasionally pronounced only in places. The right lobe is sometimes chiefly affected.

After cutting the liver, the blade of the knife is found covered with an emulsion-like fluid which contains numerous drops of fat. In well marked cases the liver burns with a bright flame.

The weight of the liver is increased, but its specific gravity is diminished, sometimes even to that of water. Fatty changes are often found in other organs.

In slight grades of the disease the hepatic cells are often filled with more or less numerous fatty granules. In marked cases the cell is filled by a single shining drop of fat; it is round and increased in size, and appears to be destitute of

membrane and nucleus. After sections are placed in turpentine or Canada balsam, the membrane and nucleus make their appearance. A few of the cells may contain crystals of fat. The fatty degeneration generally begins at the periphery of the lobules.

It was formerly held that in fatty infiltration an accumulation of large drops of fat occurred in the individual liver cells, but that these were filled with very fine fat granules in fatty degeneration. But this distinction is not real. Fatty infiltration may begin with an accumulation of fine fat granules, and the latter sometimes coalesce into a single drop of fat in fatty degeneration.

In fatty infiltration Perls found a deposit of fat in the intercellular biliary capillaries. Platen observed an accumulation of fat in the stellate cells which are scattered between the capillaries and liver cells.

The hepatic cells contain drops of fat even under normal conditions, so that the transition from the physiological to the pathological fatty liver occasionally occurs very gradually. In one case Frerichs found 78.07 per cent fat in the dried hepatic tissue. Leucin and tyrosin were also found in large quantities. In one case Frerichs found a peculiar yellow coloring matter, which differed essentially from ordinary bile pigment.

Frerichs and Perls have attempted to distinguish by chemical means the difference between fatty infiltration and degeneration. In the former the fat accumulates chiefly at the expense of the water in the hepatic cells, so that the organ becomes rich in fat, poor in water, and unchanged with regard to albuminoids. In fatty degeneration the fat forms at the expense of the albuminoids, so that the organ is rich in fat, poor in albumin, water unchanged.

Rokitansky described the so-called wax liver as a special form of fatty liver. It presents unusual firmness and brittleness and a waxy color; it is probably richer in the firmer fats (palmitin and stearin).

III. SYMPTOMS.—In many cases no symptoms are produced during life. In others we notice the mechanical symptoms arising from enlargement of the liver (a feeling of pressure and tension in the hypochondrium, perhaps real pain in the liver).

Concerning enlargement of the organ we refer to the previous section. It may be mentioned here that the lower border of the soft liver can rarely be felt distinctly.

These symptoms may be associated with functional disturbances (anorexia, eructations, vomiting, a tendency to diarrhœa and often to hemorrhoids).

IV. DIAGNOSIS.—This is not always easy. The existence of fatty liver must be assumed in "high livers" and obese individuals, even if a thick panniculus adiposus prevents the recognition of the size of the organ.

It is distinguished from cirrhosis by the less resistance of the organ to the feel, and the absence of ascites and enlargement of the spleen. In waxy liver, the resistance is greater, and the lower border can be mapped out distinctly; there is often enlargement of the spleen or œdema and albuminuria, if the kidneys are waxy.

V. PROGNOSIS.—This depends upon the causation. The disease may hasten death by interfering with digestion and nutrition.

VI. TREATMENT.—This depends upon the etiology of each individual case. Otherwise, purely symptomatic treatment is indicated.

7. Waxy Liver.

(*Amyloidosis hepatis.*)

I. ETIOLOGY.—Waxy liver is always secondary to cachectic conditions (chronic suppuration and ulceration, chronic diseases, syphilis, and intermittent fever).

Among the suppurations, tubercular affections of the bones and joints are the most important. Waxy liver, also, is not infrequently the result of abscesses of the soft parts, whether they are encapsulated or have perforated externally. Among the chronic ulcerative processes, pulmonary phthisis is the most important etiological factor. Waxy liver may be the result of the following chronic diseases: Rickets, osteomalacia, pseudoleukæmia, Bright's disease, gout, tumors (cancer, sarcoma, lymphosarcoma, fibromyoma, ovarian cysts, etc.).

Nothing positive is known concerning the connection between waxy liver and cachexia. Dickinson found that the liver was poor in alkalies; since the organism loses alkalies in suppuration, etc., he assumed that the amyloid substance is fibrin which has been deprived of alkalies (?).

The disease is almost always acquired; it may be congenital in hereditary syphilis. It is much more frequent in men than in women, and from the tenth to the fiftieth years of life.

II. ANATOMICAL CHANGES.—In well-marked cases the liver may increase to double its normal size, and its lower border is often thickened and blunted. It has been known to extend from the third rib to the crest of the ilium. The weight of the organ is increased. The capsule is usually smooth, tense, and free from adhesions. The organ presents an increased resistance when cut through. The cut surface has a peculiar lardaceous appearance. A thin section appears transparent in transmitted light. The surface looks anæmic and brownish-red; the lobular markings are indistinct. The liver is brittle and doughy, and pressure produces permanent depressions. If to the cut surface is applied a solution of iodine (potass. iodid., gr. xxx.; iodin. pur., gr. xv.; aquæ, \bar{z} iiiss.), and this is washed off after a time, we will notice a deep, reddish-brown, mahogany color of the affected parts, while the unaffected parts are light yellow or unstained. This appearance is often seen best in thin sections. The gall-bladder is often empty, or contains a mucoid fluid, more rarely greenish, inspissated bile.

When the waxy degeneration is less diffusely developed, it will be found to be confined to about the middle third of the lobules. This part is grayish, transparent, is stained of a mahogany color by iodine, while the peripheral zone is opaque (fatty changes in the cells) and the central portion deep yellow or brownish-yellow (bile staining of the cells).

In the incipient stages of waxy degeneration the diagnosis can only be made with the microscope.

In addition to waxy changes, we not infrequently find fatty degeneration of the liver cells. Amyloidosis may also develop in cirrhotic or syphilitic livers, and in the cyanotic nutmeg liver. In all such cases the waxy changes may not appear on superficial observation. They sometimes occur in small, scattered foci, for example in syphilitic cicatrices.

On microscopical examination, the amyloid parts have a peculiar, waxy gloss, are increased in size, and appear homogeneous. If microscopical sections are placed in a weak solution of iodine, the waxy parts assume a reddish-brown or mahogany color. If the section is then placed in a weak solution of sulphuric acid, hydrochloric acid, chloride of lime or zinc, the waxy parts will turn dirty violet, violet-blue, or pure blue. Boettcher recommends the following solutions: (a) gr. $3\frac{3}{4}$ iodine, gr. $7\frac{1}{2}$ potass. iodid., \bar{z} iiiss. water; (b) 2.7 ccm. sulphuric acid to 100 water.

Various aniline colors have been recently found to form valuable reagents. (a) Methyl violet (1%) stains the waxy parts bright red, the non-waxy parts assume a blue color; (b) Saffranin stains amyloid orange yellow, non-amyloid parts rose-red.

The first changes in this disease are observed in the finest branches of the hepatic artery, then in the communicating intralobular capillaries. Since the

points of communication are situated in the middle zone of the lobules, this region is affected very early. The process then spreads toward the hepatic vein, next toward the periphery of the acinus. The interlobular branches of the portal vein are finally involved. Frerichs found waxy changes even in the vessels of the capsule and the mucous membrane of the gall-bladder.

In the capillaries, the amyloid change appears as a deposit of the waxy material. The endothelium remains intact, but the lumen of the vessels is narrowed, and even occluded in places. In other places the waxy deposits are separated in the shape of shining clumps, and are converted into a sort of cellular structures, which are readily mistaken for waxy liver cells. The adjacent hepatic cells are compressed and atrophied by the waxy deposits. At the periphery of the lobules we find fatty degeneration of the cells, in their centre an unusual staining with bile pigment.

We can confirm the statements of those writers who believe that a few of the liver cells sometimes take part in the waxy change.

It has been shown that amyloid is a nitrogenous, albuminoid substance, and that leucin and tyrosin may be derived from it. It is distinguished from other albuminoids by insolubility in an acid solution of pepsin and its long resistance to decomposition.

The question as to the site of origin of the amyloid material—whether it develops in the liver or has been carried thither in the blood—is not definitely settled, although it is more probable that it is a true degeneration of the affected parts.

Waxy changes are generally found in other organs, usually in the spleen, next in the kidneys and intestines. The liver is rarely affected alone.

III. SYMPTOMS.—Waxy changes are not infrequently present in the liver without giving rise to striking symptoms. They often produce merely the mechanical disturbances observed in other forms of hepatic enlargement. Physical examination shows that the liver is enlarged, very smooth and firm, and has a sharp lower border. The spleen is also usually found enlarged.

In other cases the patients complain of functional disturbances (anorexia, eructations, vomiting, etc.), but the origin of these symptoms is determined with difficulty. If the kidneys are implicated, the urine not infrequently contains albumin, and pallor, ascites, and œdema develop. Jaundice does not occur in this disease unless the ductus choledochus is compressed by swollen and degenerated glands at the hilus.

Amyloid degeneration does not follow fully developed cachexia at once, but there has been a tendency to over-estimate the duration of the intervening stage. Cohnheim showed that the disease may develop in three months after gunshot injuries, and in Bull's case amyloid degeneration, at least in the kidneys, developed in eighteen days after an acute psoas abscess.

The disease may last for years. Death may occur from marasmus, general dropsy, œdema or inflammation of the lungs, etc.

IV. DIAGNOSIS.—In advanced cases the diagnosis is easy. Apart from the etiology we must rely upon the tense, firm consistence of the enlarged organ, which often assumes remarkable dimensions. In some cases the demonstration of waxy kidneys and spleen may be useful in differential diagnosis.

V. PROGNOSIS.—This is almost always unfavorable, although reliable authors think that recovery is possible.

VI. TREATMENT.—The prophylaxis belongs to the domain of surgery. In other respects treatment must be directed to the primary disease (phthisis, rickets, syphilis, etc.).

8. *Cancer of the Liver.*

I. ETIOLOGY.—Age exerts a certain influence. The disease is most frequent from the fortieth to the sixtieth years, especially in women at the menopause. It is rare in the first two decennia, but Siebold reported a case in a new-born infant.

Cancer of the liver is more frequent in women, because it is often secondary to cancer of the breast, uterus, or ovaries.

A few observations indicate that heredity is an occasional etiological factor. The disease is rarely observed in the tropics.

Injury is sometimes mentioned as a cause. Reliable authors include gall-stones among the traumatic factors. At all events gall-stones are often found in cancerous livers, but this may perhaps be explained as an accidental complication or as a secondary development of the calculi on account of occlusion of the bile-ducts by the cancer.

Cancer of the liver may be primary or secondary. In rare cases primary cancer gives rise to secondary development of cancer in other organs. Secondary cancer of the liver usually follows the development of tumors in organs which are connected with the portal vein either directly or by means of collateral branches (uterus, rectum, stomach, etc.). Cancer of remote parts (eye, brain, etc.), may also give rise to secondary deposits in the liver. In some cases the growth spreads to the liver by contiguity, for example, from the stomach.

Secondary cancer of the liver is much more frequent than the primary form.

III. ANATOMICAL CHANGES.—Cancer of the liver may be circumscribed or infiltrated. In the former variety we find sharply defined tumors, in the latter a diffuse infiltration which often passes very gradually into the healthy tissues.

The nodules of cancer may vary from the size of a pin's head to that of a child's head. In primary cancer a single growth or a small number is usually present, in secondary cancer we find a large number of tumors, sometimes more than one hundred. If but a single tumor is present, it generally involves the right lobe.

The liver sometimes increases more than sixfold in weight. It may extend from the third or even the second rib to beneath the anterior superior spinous process of the ilium. The hepatic substance proper diminishes as the cancerous masses increase.

Small nodules within the liver will only be recognized on section. If they extend to the capsule, they often project beneath it above the surface of the liver, and the prominences frequently present a plate-shaped depression (umbilication). Cancerous nodules are sometimes found on the opposite side of the diaphragm or abdominal wall, to a certain extent as the expression of a local infection.

Virchow regards the umbilication as the result of fatty degeneration of the cancer cells and of cicatricial formation in the central, oldest portion of the tumor. Cicatrization of the entire nodule is prevented by the new formation of cancerous tissue at the periphery of the cancer.

Frerichs observed umbilication in young nodules, and attributes it to the fact that the connective tissue at the centre of the nodule is more contractile than that at the periphery and the amount of cancer juice is less.

Primary circumscribed cancer forms a rounded tumor which usually projects somewhat above the cut surface, has a creamy cancer juice, and contains fatty, occasionally cheesy or hemorrhagic foci. Its periphery

may be sharply defined, or in places it may pass gradually into the healthy tissue. The adjacent hepatic tissue is compressed, and has a pale or brownish-red color.

The tumor sometimes breaks into adjacent vessels, such as the hepatic veins, portal veins, finally into the biliary canals. Metastases are rarely found in remote organs.

In primary infiltrated cancer of the liver the appearances are somewhat like those of cirrhosis. The liver is traversed by broad bands of connective tissue, and its surface is nodular. Within the connective-tissue bands are little islets of cancer cells. No retrogressive metamorphoses occur except fatty degeneration. The infiltration sometimes extends to the wall of the gall-bladder, but proliferation into the vessels or metastases to other organs are not observed.

In both forms, the portal glands are often implicated and may produce serious symptoms by pressure on the excretory ducts or portal vein.

Naunyn showed that the cancer develops from a proliferation of the epithelium cells in the biliary canals and that these dilate, in places, into alveoli. Some writers believe that the liver cells are also converted into cancer cells, or that both modes are combined. The tumor is nourished by the hepatic artery.

In secondary cancer of the liver, the tumor is almost always circumscribed. Its histological character depends upon that of the primary growth (alveolar, scirrhus, colloid, cystic cancer, fungus hæmatoides, cylindrical and pavement cell cancer).

The cancer germs may be transported to the liver through the portal vein, hepatic artery, or the lymphatics. In some cases, corpuscular elements are probably carried to the liver as emboli, proliferate and affect the adjacent tissue.

Secondary cancer may also proliferate into the biliary canals, portal and hepatic veins, and the gall-bladder.

Secondary cancer of the liver is also an epithelial new-formation. Its cells are derivatives of hepatic cells, its stroma is derived from the capillaries and surrounding connective tissue; it is nourished by the hepatic artery.

Cancer of the liver is a frequent disease. In the Vienna General Hospital one case occurred to three hundred and twenty-two cases of other internal diseases.

III. SYMPTOMS.—In not a small number of cases, cancer of the liver is unrecognized, because the tumor, on account of its small size and localization, produces neither mechanical nor functional disturbances.

In other cases, the symptoms are concealed by those furnished by other organs (cancer of the stomach, rectum, etc.).

In some cases there is marked ascites, whose cause is unrecognized until the autopsy. Or the patients may die with symptoms of ichorous or hemorrhagic pleurisy, and the autopsy reveals cancer of the liver and secondary deposits on the pleura. Finally, the symptoms may consist of undefined malaise and increasing marasmus.

The chief symptoms consist of mechanical and functional disturbances of the liver (enlargement, nodular surface and tenderness of the liver, and jaundice).

The liver may attain remarkable dimensions in this disease. An important feature is the increase in size under observation. In Farre's case the organ increased five pounds in weight within ten days. It presents respiratory displacement until it has become so large as to be impacted and immovable in the abdomen. In rare cases, atrophy and diminution of the size of the liver are observed (three times in thirty-one of Frerichs' cases).

An important symptom is the recognition of prominences on the surface of the liver. Special attention should be paid to the lower border of the organ. Frerichs also recommends careful examination along the inner border of the rectus abdominis. The tendinous insertions along the belly of the muscle may be mistaken for nodules on the liver. The prominences are sometimes visible to the eye. They are usually hard, very rarely fluctuating. In a very emaciated patient, I was able to detect the umbilication of the nodules, but this is very exceptional. The nodules also present respiratory displacement (unless adhesions to the diaphragm or anterior abdominal walls have formed), and sometimes cannot be felt except after deep inspirations. Peritonitic friction murmurs can sometimes be felt and heard (perihepatitis). The enlarged liver sometimes presents pulsating movements, which are evidently conveyed from the aorta. These movements consist of a simple lifting and falling of the organ.

Pain is rarely absent; it is sometimes spontaneous, sometimes produced only on pressure. It is sometimes limited to the hepatic region, or it may radiate to the sacrum, right shoulder, or arm. In some cases there is merely a feeling of pressure or tension in the right hypochondrium. In one case, Henoeh observed anæsthesia of the right half of the body.

Jaundice is an important, though not a constant symptom. It depends chiefly on compression of the larger bile-ducts by the tumor or degenerated lymphatic glands. It constantly increases in intensity, and the skin may assume a blackish, greenish-yellow color. Bronzed skin is occasionally noticed.

As a rule, the general nutrition is soon impaired in a striking degree. The patient emaciates, the skin has a grayish-yellow or cachectic color, the strength diminishes more and more. A few cases have been reported in which the nutrition of the body remained good until death.

In very rare cases, there is swelling of the jugular lymphatic glands immediately over the sternal portion of the left clavicle, as the result of cancerous infection through the thoracic duct. The inguinal glands are also occasionally swollen and indurated.

The bodily temperature may remain normal or even subnormal (34° C.). In other cases there are temporary rises of temperature, occasionally of a hectic or intermittent type. Some cases have been reported in which the disease ran a rapid, febrile course.

At first, the pulse is not infrequently slow; later it becomes frequent, soft, occasionally irregular. Consciousness may remain intact to the last moment. In other cases, delirium occurs (particularly towards the close of life), either as the result of inanition or febrile movement.

Many patients complain of obstinate insomnia, others of violent pruritus.

The appetite is usually lost, thirst is often increased. The tongue has a grayish or brownish coating, and not infrequently is remarkably dry. Vomiting is a frequent symptom. At times it cannot be checked, if the pylorus is compressed by the tumor in the liver.

Enlargement of the spleen is generally absent. Even if the portal vein is compressed, the marasmus prevents passive congestion of the spleen. In rare cases, it is enlarged as the result of cancerous proliferation.

Ascites develops not infrequently. It may be merely the expression of general marasmus, the result of secondary carcinomatous degeneration

of the peritoneum, of chronic peritonitis or of occlusion of the portal vein. The transudation may have an amber yellow or a hemorrhagic color; its specific gravity varies from 1.009 to 1.014, its percentage of albumin from one to four. Transitions to an inflammatory exudation are sometimes noticed.

The ascites may give rise to dilatation and sinuosity of the subcutaneous abdominal veins.

The bowels are generally constipated; in cases of jaundice, the stools may be destitute of bile.

The urine is usually diminished and dark colored; the amount of indican may be increased. With increasing marasmus traces of albumin may appear in the urine.

On examination of the blood, it is often found that the red blood-globules are scanty, very pale, and present striking variations in size and shape (poikilocytosis). The white blood-globules may be somewhat increased in number. Not infrequently we find an unusual number of small, colorless clumps of protoplasm (elementary granules). These changes are the result of the general marasmus rather than of the cancer.

The disease may run an acute, subacute, or chronic course. Bamberger reported a case in which the disease presented symptoms similar to those of acute hepatitis, and terminated in eight weeks. Frerichs observed a fatal termination at the end of four weeks. In other cases, the disease lasts several months, one or two years, perhaps even longer. The average duration is probably four to five months from the appearance of the first symptoms.

Death may occur from increasing marasmus, from suffocation (the result of ascites, hydrothorax, size of the tumor), extensive bronchitis or pneumonia. Unexpected accidents occasionally prove fatal. Thus, a cancer nodule at the surface of the liver may break down, and give rise to diffuse peritonitis, or profuse hemorrhage occurs from a degenerated nodule. Fatal syncope has been observed when the patients attempted to sit up.

In one of my cases, the cancer became adherent to the anterior abdominal wall, and perforated externally.

IV. DIAGNOSIS.—The diagnosis is often extremely difficult. Perhaps jaundice is the sole symptom. If other causes can be excluded, if rapid emaciation occurs and the patient is advanced in years, a probable diagnosis of cancer of the liver may be made.

In other cases jaundice is absent, but an uniform enlargement of the liver is demonstrable. In such cases we must exclude other causes of enlargement of the liver, and consider the age of the patient, rapid marasmus, and tenderness of the liver.

Tumors of adjacent organs (pylorus, omentum, colon, kidneys, pancreas, coprostasis in colon, etc.) may be erroneously attributed to the liver.

Tumors of the liver move on respiration, those of the other organs do not. Functional disturbances of the various organs must also be taken into consideration. In cancer of the pylorus, the position of the tumor changes according to the distention of the stomach, and the tumor is sometimes separated from the liver by a tympanic zone. The diagnosis may be very difficult if the tumor becomes adherent to the liver. Omental tumors are often characterized by great mobility and low position. In cancer of the colon we find the signs of intestinal stenosis.

The distended gall-bladder may be mistaken for an hepatic tumor, but the former is usually smooth and has the characteristic pear shape.

If it has been rendered certain that the tumor is situated on the liver, the diagnosis of cancer is favored by advanced age, marasmus, and firm consistence of the growth. Fluctuation is noticed in hepatic abscess and echinococcus. In multilocular echinococci the spleen is usually enlarged. Cirrhosis and tight-laced fissure of the liver may also be sources of error. In the former, great weight must be attached to alcoholic excesses and enlargement of the spleen, in the latter to the presence of an external fissure from tight-lacing, and the absence of pain and cachexia.

V. PROGNOSIS.—The disease is always fatal.

VI. TREATMENT.—The treatment is purely symptomatic.

APPENDIX.

a. Sarcoma of the liver is usually metastatic; it is very rarely primary. It is generally multiple, and may give rise to considerable enlargement of the organ. Its structure depends upon that of the primary tumor, but osseous tissue never forms in the metastatic hepatic growths. Melanosarcoma appears as circumscribed nodules or as a diffuse infiltration. In the latter event, the liver may present a marbled appearance. The pigment is situated partly within, partly outside the tumor cells.

The clinical symptoms are the same as those of cancer of the liver. In one case, Leopold detected a vascular murmur over the liver, and this he attributed to the abundance of blood-vessels in the tumor. Melanuria has been observed in melanosarcoma. The starting-point of the latter is found particularly in tumors of the eye.

b. Adenoma of the liver cannot be distinguished clinically from cancer. We usually find multiple tumors which may attain the size of an apple.

c. Fibroma, lipoma, glioma, myxoma, cysts, and angioma of the liver possess merely an anatomical interest.

9. *Echinococcus of the Liver.*

I. ETIOLOGY.—Among all the internal organs, the liver is the most frequent site of echinococcus vesicles.

In man, these form the vesicular condition of the *tænia echinococcus*. The mature tape-worm itself has not been found in the human species. It occurs in dogs and other varieties of canidæ (wolf, jackal). In dogs it generally inhabits the upper part of the small intestines. The mature *tænia* is four mm. long, and consists generally of a head and four links, the last one being sexually ripe (Fig. 61). The head is furnished with a rostellum, four sucking disks, and a double row of hooks (thirty to fifty).

The ova are always derived from dogs, and we find accordingly that echinococcus vesicles occur most frequently in those regions in which dogs come in closest contact with human beings.

The ova may be conveyed to man by allowing dogs to lick the individual's face. According to some writers, drinking-water which has been fouled by the fæces of dogs may act as the source of infection.

Echinococci are especially frequent in Iceland, because the inhabitants live in close contact with their domestic animals, and strict attention is not paid to the laws of cleanliness. A similar frequency, though not to such a marked extent, is also noticed in various parts of other countries.

Echinococci are rarely found in children and old people.

II. ANATOMICAL CHANGES.—The path by which the ovum of *tænia echinococcus* makes its way from the intestinal canal to the liver is not known with certainty. It is supposed that the gastric juice removes its membranes, and that, by means of its hooklets, it enters the radicles of the portal vein, whence it is carried, by the blood current, to the liver. Echinococci appear in the liver in two forms, viz., *echinococcus unilocularis* (most frequent variety), and *echinococcus multilocularis* (rarer variety).

Echinococcus unilocularis forms, as a rule, around vesicle filled with fluid, and which is sometimes as large as a man's head. There is usually but a single vesicle, more rarely several. The right lobe of the liver is the favorite site.

The vesicle may be situated deep in the parenchyma or near the surface. If it extends to the capsule, the latter is often thickened and opaque, occasionally adherent to adjacent parts. The vesicles are sometimes connected with the liver by a sort of pedicle.

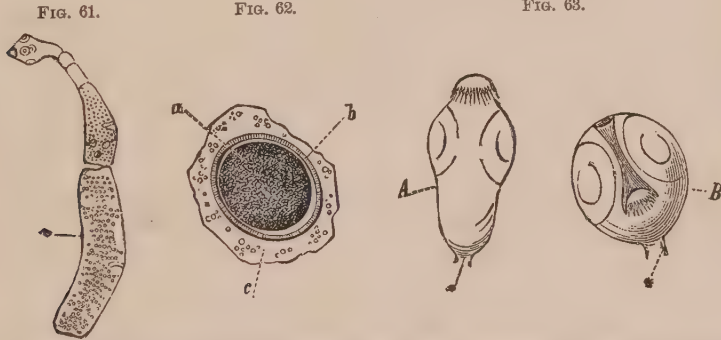


FIG. 61.—*Tænia echinococcus* from the intestine of the dog. The last link mature and about to be cast off spontaneously. Enlarged 8 times.

FIG. 62.—Ovum of *echinococcus* with albumin covering. *a*, embryo with six hooklets; *b*, shell of ovum; *c*, covering of albumin. Enlarged 600 times.

FIG. 63.—Heads of *echinococci*. *A*, with protruded rostellum; *B*, with retracted rostellum.

The liver is necessarily increased in size. In some cases, it has extended from the second rib to the spine of the ilium, and displaced the lungs, heart, stomach, intestines, and spleen. Vesicles are also found not infrequently in other organs.

The *echinococcus* vesicle proper is surrounded by a fibrous capsule—the product of reactive inflammation of the adjacent parenchyma. It is vascular, and usually has a thickness of a few millimetres.

The vesicle itself consists of an external membrane, of a milk-glass color, which, when punctured, gives exit to a clear, amber-yellow fluid. If the vesicle is incised, the free edges roll inward (vide Vol. I., Fig. 86), and a granular mass is noticed on the inner surface of the wall of the vesicle (parenchyma layer). If the fluid is allowed to stand, a sediment is deposited similar in character to the parenchyma layer.

In many cases, the vesicles contain “daughter cysts,” and these others in their turn. The daughter cysts are sometimes so numerous that they compress one another, and even rupture the mother cysts. In one case, Allen counted nearly eight thousand daughter cysts.

Under the microscope, the wall of the *echinococcus* is found to have a

laminated structure (Vol. I., Fig. 87). On the parenchyma layer are found brood capsules, and on these echinococcus heads or scolices (vide Fig. 63). The head is connected with the parenchyma layer by a thin pedicle. In other cases, it is torn off, and floats free in the fluid contents of the vesicle. Sterile echinococci (destitute of heads) are rarely found.

Luecke attempted to show that the membrane proper is formed of a chitin-like substance. The quantity of fluid in the vesicle is sometimes very considerable (twenty-eight pounds in Tommasini's case). Its reaction is usually neutral, more rarely alkaline, still more rarely acid, and its specific gravity varies from 1.007 to 1.015. A characteristic feature is the absence of albumin, and the presence of succinic acid (brown color on the addition of a dilute solution of ferric chloride), though this is not constant. Grape sugar, leucin, inosit—a casein-like substance—and considerable sodium chloride have also been found in the fluid.

The parenchyma immediately adjacent to the vesicles is usually in an atrophic condition. If several vesicles have developed in the liver, the greater part of the parenchyma may be destroyed. Hyperplastic changes have been repeatedly found in other parts of the organ.

The vesicle occasionally undergoes suppuration or destruction. In the former event, an abscess forms, in which the resistant hooklets alone remain to indicate the origin of the suppuration. In the latter event, the capsule is converted into a thick, cartilage-like callosity, which often undergoes calcification. The contents then undergo thickening, and are converted into a putty-like mass, in which are found hooklets, cholestearin, hæmatoidin, bile pigment.

Echinococcus multilocularis is much less frequent than the unilocular variety. In 1878, Bæumler collated thirty-seven cases, and perhaps a dozen have been reported since that time. In Iceland, in which unilocular echinococcus occurs so frequently, not a single example of the multilocular variety has been observed.

It is situated generally in the right lobe of the liver, and forms a hard, many-chambered tumor, the individual cavities being filled with gelatinous contents. These cavities correspond to echinococcos vesicles and sometimes contain scolices or hooklets. The internal wall is usually ulcerated; in places we notice calcification. The spleen is almost always enlarged.

The development of multilocular echinococcus is exogenous, *i. e.*, new vesicles form outside of the mother vesicle. This is facilitated by the circumstance that it develops within pre-existing canals (lymphatics, biliary canals, even the blood-vessels).

III. SYMPTOMS.—Unilocular echinococci of small dimensions which are situated within the liver may remain unrecognized during life. In some cases, vague symptoms are observed, such as eructations, vomiting, digestive disturbances.

Notable disturbances will only be produced if the liver has increased considerably in size, if the vesicles are situated superficially, or they compress the blood-vessels or biliary ducts, or rupture into adjacent organs and then appear externally.

The liver is sometimes enlarged to such an extent that hepatic dulness extends from the second rib to the spine of the ilium. This will produce compression of the right lung and displacement of the heart, and thus give rise to dyspnoea. This is increased still further by interference with the movements of the diaphragm, and atrophy of the muscle from pressure.

The right hypochondrium and thorax are often visibly distended.

Vesicles which project above the surface of the liver not infrequently give rise to an abrupt convexity which projects into the right thorax. The line of dullness then extends high upwards, usually in the lateral region of the right thorax, while it makes a sudden fall anteriorly and near the spine. Marked enlargement of the liver causes a feeling of tension, fulness, and even pain in the right hypochondrium. It may also cause weakness and paræsthesia of the lower limbs and œdema.

In some cases prominent vesicles can be reached with the hand. They present respiratory movements unless prevented by adhesions, or unless the increase in the size of the liver is so considerable that the position of the liver no longer changes.

The vesicles are usually felt as tense, elastic tumors, and not infrequently present fluctuation. If the waves of fluctuation are very small, the so-called hydatid tremor is produced. Some authors claim to have heard a peculiar buzzing on auscultation of the vesicle.

Hydatid thrill was absent in more than half of Frerichs' cases. On the other hand, it has been observed in ascites and ovarian cysts. It is especially distinct if the wall of the vesicle is not too tense and contains daughter vesicles. Kuester recently described a case in which two vesicles were situated alongside of one another and gave rise to very distinct thrill. It is best observed when the vesicle is held, during percussion, between the thumb and index finger of the left hand. Davaine recommended placing three fingers of the left hand on the vesicle and percussing the middle one.

The vesicle sometimes ruptures into adjacent organs. Rupture into the pericardium is usually followed by rapidly fatal pericarditis. Similar results follow rupture into the pleura, although cases have been reported in which the pleurisy was operated upon, the vesicles removed, and recovery ensued. If the rupture is preceded by adhesion of the right lung to the diaphragm, the pulmonary parenchyma is infiltrated with the echinococcus vesicles. The vesicles are sometimes expectorated, or scolices or hooklets are found in the sputum. The latter is purulent, bloody, or putrid, not infrequently contains bile and hæmatoidin crystals. Leyden recently observed an ochre-yellow sputum, containing numerous little masses, which consisted chiefly of needles of bilirubin. Rupture into the stomach or intestines is usually associated with sudden, violent pains, and vesicles are found in the vomited matters or stools. Rupture into the bile-ducts produces jaundice and the symptoms of biliary colic. If the bile enters the vesicles, the echinococci usually die in a short time. After rupture into the inferior vena cava rapid death from suffocation may occur, as soon as the vesicles pass through the right heart into the pulmonary artery. Rupture into the hepatic veins is followed by suppuration, pyæmic conditions, or profuse hemorrhage. Rupture through the integument is often preceded by inflammatory changes in the skin. If the vesicle ruptures and its contents enter the peritoneal cavity, the signs of peritonitis acutissima usually develop. Rupture generally follows injury or excessive exertion.

Suppuration of echinococcus vesicles is followed by symptoms similar to those of hepatic abscess—chills, hectic fever, sweats, emaciation.

The vesicles sometimes compress the bile-ducts or portal vein, and give rise to incurable jaundice or portal occlusion.

Multilocular echinococcus generally forms a very firm, rarely fluctuating tumor, which is sensitive on pressure. The spleen is generally enlarged, and there is a purulent or serous exudation in the peritoneal

cavity. As a rule, we find intense jaundice, and often gastric and intestinal hæmorrhage. Ott and Niemeyer mention cutaneous œdema as an almost constant symptom. The disease is a protracted one (eleven years in Griesinger's case).

IV. DIAGNOSIS.—If the liver is enlarged and its surface contains prominences, the disease must be differentiated from cirrhosis, abscess, syphilis, and cancer of the liver.

In cirrhosis, alcoholic excesses, ascites, and enlargement of the spleen are characteristic features.

In abscess, we find the symptoms of internal suppuration—chill, hectic fever, sweats, and increasing emaciation.

In syphilis, we must look for specific changes in the skin, mucous membranes, bones, and genitalia.

Cancer occurs in old, marantic individuals. The presence of hydatid thrill favors the diagnosis of echinococci. An auxiliary diagnostic measure is puncture with a fine trocar. A clear fluid escapes, which is free from albumin, contains a large amount of sodium chloride, and sometimes succinic acid.

Projecting vesicles on the lower surface of the liver may be mistaken for tumors of the gall-bladder, but the latter are pyriform in shape. If movements are conveyed to the tumor by the aorta, it is possible to mistake it for aortic aneurism; the movements in the former are a simple lifting and falling.

If the tumor projects into the right thoracic space, it is distinguished from pleurisy by the course of the upper line of dullness, which is highest, in echinococcus, in the lateral region. In echinococcus the heart is pushed upwards, in pleurisy to the side. If necessary, an exploratory puncture may be made.

Multilocular echinococcus is mistaken most frequently for cancer of the liver. Importance should be attached to the existence of enlargement of the spleen in echinococcus.

V. PROGNOSIS.—The prognosis is not very favorable, because the echinococcus has a constant tendency to increase in size. In addition, the prognosis is often rendered worse by various complications.

VI. TREATMENT.—Prophylaxis should be carefully considered. Direct contact with dogs should be avoided, and the food should be kept clean.

If the presence of unilocular echinococcus in the liver is ascertained with certainty, the parasite should be removed as soon as possible. This is a matter of surgical interference, the details of which cannot be considered here. The methods employed are: *a.* simple puncture; *b.* puncture and aspiration; *c.* puncture and injection of irritating fluids; *d.* double puncture and incision; *e.* application of caustic paste, with subsequent incision; *f.* sewing the wall of the vesicle to the abdominal wall, and subsequent incision; *g.* electrolysis.

In multilocular echinococcus, the treatment is purely symptomatic.

APPENDIX.

Pentastomum denticulatum, *cysticercus cellulosæ*, and *psorospermia* have also been found in the liver, but possess no clinical interest.

10. *Changes in the Position of the Liver.*

Changes in the position of the liver are congenital or acquired.

a. Among the former is inversion of the viscera, the liver being

situated in the left, the spleen in the right hypochondrium. The position of the other viscera also is usually reversed.

In diaphragmatic hernia, the liver has been found partly in the pleural cavity.

b. Acquired displacement of the liver is often the result of thoracic and abdominal diseases. The organ is pushed downwards in thoracic affections, upwards in abdominal diseases.

Wandering or migrating liver requires more careful attention. It occurs almost exclusively in multiparous women who have passed the age of 30 years.

The organ is situated unusually low in the abdomen, on account of the yielding of the suspensory ligament. In marked cases, the upper border of the liver is below the lower border of the thorax. In a few cases, the tense suspensory ligament could be felt as a thin strand. The liver is situated mainly in the right side of the abdomen, and is usually freely movable. An important diagnostic feature is the fact that the tumor can be pushed upwards into the normal position of the liver, and thus causes disappearance of the tympanitic zone which had previously been present between the upper border of the liver and the thorax. The patients were often the first to notice the tumor, complained of a feeling of tension and dragging, and became nervous and hysterical. Barbarotto's patient complained of pain in the right shoulder.

Very little is known concerning the causes of this condition. In some cases, it is said to have been the result of heavy lifting or tight lacing. It appears to be favored by pregnancy and relaxation of the abdominal walls, especially if associated with congenitally unusual length of the suspensory ligament.

The diagnosis is not easy. Frerichs mentions a case in which cancer of the omentum reproduced the exact contours of the liver. Legg calls attention to the possibility of mistaking it for renal, ovarian, and uterine tumors. In making a diagnosis of wandering liver, we should pay attention to the shape of the tumor, its respiratory mobility, the absence of hepatic dulness in the normal situation, and its re-appearance after reposition of the tumor.

11. *Changes in the Shape of the Liver.*

(Tight-laced Fissure.)

Changes in the shape of the liver are congenital or acquired. The former possess no clinical significance; and of the latter, tight-laced fissure alone remains for consideration.

It is found in women who corset tightly. A horizontal, more or less deep furrow is thus produced, partly by the lower ribs, partly by the direct action of the tight articles of clothing. It generally affects only the right lobe, more rarely also the lower part of the left lobe. In this situation, the hepatic tissue is atrophic, and the capsule is thickened. The constricted portion of the liver is usually club-shaped, its lower border being blunted. Fibrous hyperplasia may extend from the fissure into the constricted portion of the liver, and impart to it an increased consistence.

This condition possesses a clinical interest, in so far as the constricted portion may be mistaken for a tumor of other organs. This is especially apt to occur if the constricted part is very movable, hard, and prominent,

and if a loop of intestine has entered the fissure, so that the lower part is separated from the area of hepatic dulness proper by a tympanitic zone. But on careful examination the fissure can generally be felt, and if a loop of intestine is situated in it, firm percussion should be exercised, whereupon the previous tympanitic note becomes dull.

C. DISEASES OF THE BLOOD-VESSELS OF THE LIVER.

1. *Stenosis and Occlusion of the Portal Vein.*

I. ETIOLOGY.—In the majority of cases, this condition is the result of thrombosis, since even if other causes have been at work, they will generally be followed by thrombosis. Hence pylethrombosis and portal occlusion are almost identical terms.

Portal thrombosis may develop if the circulation is abnormally slow, although fatty degeneration of the endothelium of the vessel is probably a necessary element of coagulation of the circulating blood. This has been observed in phthisis and marantic conditions in general.

The disease occurs much more frequently from compression of the portal vein, especially in diseases of the liver. It is not infrequent in cirrhosis, the intra-hepatic branches of the vessel being first affected, and the thrombus then passing backwards into the trunk. It occurs less frequently in cancer, still less frequently in abscess of the liver.

The portal vein is sometimes compressed as the result of unusual dilatation of the bile-ducts, enlargement of the glands at the hilus, or tumors of adjacent abdominal organs (stomach, duodenum, etc.).

Finally, the portal vein may be compressed by retracting cicatrices in chronic peritonitis.

II. ANATOMICAL CHANGES.—Thrombi of the portal vein sometimes occlude the vessel entirely, sometimes they merely line its wall. The thrombosis may affect the trunk, the intra-hepatic branches, some of the radicles of the portal vein, or all of them. In recent cases the clots are brownish-red; older ones lose their color, are firm, occasionally of a cheesy yellow color. Organization of the thrombus occurs much less frequently than is generally believed; the vein may then be converted into a solid fibrous strand.

The walls of the vessel may undergo thickening, fatty degeneration, or calcification.

When the occlusion is limited to a few hepatic branches, the liver presents a peculiar lobulation, which must be regarded as the result of secondary proliferation of connective tissue.

III. SYMPTOMS.—The occlusion of a few intra-hepatic branches presents no symptoms during life. But if the greater part of the intra-hepatic branches, or the trunk of the vessel itself, is affected, very characteristic symptoms of stasis are produced.

The spleen is enlarged, except in those cases in which the capsule is very thick or the organ is waxy, or the enlargement is prevented by considerable losses of blood. Ascites is an almost constant symptom. It is apt to return very quickly after the fluid has been removed by puncture.

Disturbances of digestion (anorexia, nausea, vomiting, etc.) are prominent. If the venous congestion increases, hæmatemesis and enterorrhagia may develop. Jaundice is rare, even in complete occlusion

of the portal vein. The subcutaneous abdominal veins are dilated, as in cirrhosis.

The disease may last for months and even years. Death generally results from marasmus.

IV. DIAGNOSIS.—Portal occlusion is distinguished with difficulty from cirrhosis of the liver. In making the differential diagnosis it should be remembered that the liver is hard and nodular in cirrhosis, and that the disease is often the result of alcoholic excesses, and that, in portal occlusion, ascites is apt to return very quickly after puncture.

Portal occlusion may also be mistaken for tuberculosis and carcinoma of the peritoneum, and for chronic serous peritonitis.

V. The prognosis is always unfavorable. The treatment is similar to that of cirrhosis of the liver.

2. *Suppurative Inflammation of the Portal Vein.*

Suppurative Pylephlebitis.

I. ETIOLOGY.—Except in rare cases the disease is secondary. The protected position of the portal vein renders it quite impregnable to traumatism, unless the latter originates in the gastro-intestinal tract. Two cases have been reported in which a fish-bone and a piece of wire which had been swallowed gradually penetrated the walls of the portal vein, and set up a suppurative inflammation.

Secondary suppurative pylephlebitis generally follows inflammatory and ulcerative processes in those organs in which the radicles of the portal vein have taken their origin. These include perityphlitis, paratyphlitis, intestinal ulcers, dysentery, ulceration of the dilated hemorrhoidal veins, operations on the rectum, gastric and duodenal ulcers, ulcerated cancer of the stomach and duodenum, abscess of the spleen, inflammation of the pancreas, omentum, or mesentery, encapsulated peritonitis, cheesy and suppurating retroperitoneal glands, ovarian cysts.

The disease takes its origin less frequently in the liver itself. Thus, gall-stones or obstinate stasis of bile from other causes give rise not infrequently to suppurative inflammation of the bile-ducts. This extends to adjacent parts, and thus implicates the intra-hepatic branches of the portal vein. In rare cases, this is also observed in abscess or echinococcus of the liver, after perforation into the branches of the portal vein. In the new-born, pylephlebitis sometimes follows inflammation of the umbilicus, but Schuëppel has shown that the umbilical vein is normal in some of these cases.

II. ANATOMICAL CHANGES.—Suppurative inflammation of the portal vein may affect a few of its radicles or intra-hepatic branches, or the trunk of the vessel, or the greater part of the entire portal system. The inflammation often extends uninterruptedly from the radicles to the peripheral intra-hepatic branches; in other cases unaffected and inflamed portions alternate with one another. Puriform, infectious masses are very apt to separate from the radicles, pass into the intra-hepatic branches, and there set up fresh inflammation.

On opening the portal vein and its branches, the lumen is found to be filled with a smeary, grayish-red, puriform, ichorous mass. The affected vessel is thickened and hard to the feel.

The tunica intima is opaque, wrinkled, and in places presents losses of substance and ulcerations (pylephlebitis ulcerosa). The media and

adventitia are thickened, injected, succulent, and often infiltrated with pus.

The liver and spleen are usually enlarged. The liver often contains numerous foci of pus, the result of extension of inflammation from the vessels to the adjacent parenchyma. In some cases the pus enters the inferior vena cava through the hepatic veins and gives rise to embolic abscesses in the lungs. This may be followed by abscesses in the spleen, kidneys, etc.

III. SYMPTOMS. —Purulent pyelephlebitis is almost always attended with pain in the epigastrium and right hypochondrium. It is either spontaneous or produced by pressure in this region.

As a rule, the liver is tender and enlarged. The spleen is also enlarged, probably as the result of the general infection. Jaundice is almost always present, and is sometimes very intense. It must be regarded as chiefly hæmatogenous, since the urine often contains no biliary coloring matter and the fæces retain the bilious character.

Fever is almost always present. As a rule, numerous chills occur, followed by an exacerbation of temperature. The chills and fever sometimes recur at such regular intervals as to arouse the suspicion of intermittent fever.

The general condition fails very rapidly. The patients are apathetic, somnolent, and delirious. In addition, anorexia, vomiting, and diarrhœa accelerate the loss of strength. Signs of blood dissolution sometimes appear (hemorrhages upon the skin and mucous membranes). Symptoms of pyæmic suppuration may also make their appearance (bloody sputum, thoracic or peritoneal dulness, swelling of the joints, etc.).

The disease often lasts only a few days, in other cases two weeks; it is occasionally prolonged for six weeks. It always terminates fatally.

IV. DIAGNOSIS.—The disease may be mistaken for the following conditions:

(a) *Abscess of the liver.* In this affection enlargement of the spleen and diarrhœa are absent, and the causes are often different.

(b) *Biliary colic.* Enlargement of the spleen and diarrhœa are wanting, the stools are often discolored, the urine contains biliary coloring matter, the patients do not emaciate rapidly.

(c) *Catarrhal jaundice.* The urine contains biliary coloring matter, the fæces are deficient in bile, there are no chills.

(d) *Intermittent fever.* Jaundice is absent, the chills recur more regularly, and are rapidly relieved by quinine.

V. The treatment is purely symptomatic.

3. Aneurism of the Hepatic Artery.

Five cases of this affection have been reported. The symptoms were: A pulsating tumor to the right of the linea alba, attacks of pain (similar to biliary colic or gastralgia), jaundice, hæmatemesis, and enterorrhagia.

Chiari recently described a case of aneurism of the cystic artery, which ruptured and led to fatal hæmorrhage.

PART VI.

DISEASES OF THE PANCREAS.

Diseases of the pancreas possess very little clinical importance, and, as a rule, lesions of the organ are found unexpectedly at the autopsy. Functional disturbances may be absent, even if the gland is entirely destroyed. A number of cases have been observed in which recovery occurred after destruction of the gland.

There is not a single symptom which positively indicates disease of the pancreas. Although the gland performs an important part in the digestion of albuminoids, carbo-hydrates, and fats, the rest of the digestive apparatus is able to assume its function.

We will confine ourselves to a few hints with regard to pancreatic diseases.

1. *Hemorrhage*.—Extravasations into the pancreas usually possess but little importance, and are found associated with hemorrhages into other organs in conditions of stasis and dissolution of blood (scurvy, pernicious anæmia, etc.). Zenker has recently called attention to extensive hemorrhages which may prove rapidly fatal. The development of this condition appears to be favored by fatty degeneration of the gland. Zenker explains the sudden death as the result of shock; Friedreich attributes it to pressure on the semilunar ganglion and solar plexus. The diagnosis is impossible.

I recently observed a case of ileus which was caused by extensive hemorrhage into the pancreas. The organ was enlarged to such an extent as to occlude the duodenum by pressure.

2. *Pancreatitis*.—This may be acute or chronic. The former has a tendency to the formation of pus, the latter to an increase of the interstitial tissue. The symptoms (pain in the upper part of the abdomen, vomiting, anorexia, fever, loss of strength, etc.) are not of such a character as to permit a diagnosis, though Oppolzer recognized the disease in one case.

3. *Cancer*.—The disease begins generally beyond the age of 40 years, and is nearly twice as frequent in men as in women. It is the most frequent of all diseases of the pancreas. Scirrhus is the most frequent form; medullary cancer is less frequent. A few cases of cylindrical epithelial cancer and colloid cancer have also been reported.

The head of the gland is usually involved, more rarely the middle portion, most rarely the tail. The cancer may be primary or secondary; the latter may be metastatic, or conveyed per contiguitatem.

Symptoms may be entirely wanting. In several of my cases, the symptoms (jaundice, ascites, enlargement of the spleen) pointed to an affection of the liver. The most positive sign is the demonstration of a tumor which is situated transversely across the spine, a little above the umbilicus, and which cannot be attributed to the stomach, liver, intestines, spleen, or lymphatic glands. The tumor sometimes receives pulsations from the underlying aorta, or it compresses this vessel, and produces murmurs of stenosis.

APPENDIX.

Diseases of the mesenteric and retroperitoneal glands are usually secondary. For example, the mesenteric glands are affected in the majority of intestinal diseases. Primary changes develop more frequently in the retroperitoneal glands. Cancer or lymphosarcoma may develop in them, and occasionally attain very large dimensions. They may be felt as nodular tumors through the abdominal walls, vagina, and rectum. They produce pressure on the stomach, intestines, aorta, kidneys, ureters, bladder, and nerves. Small growths are apt to be mistaken for tumors of the different viscera.

PART VII.

DISEASES OF THE PERITONEUM.

1. *Inflammation of the Peritoneum. Peritonitis.*

I. ETIOLOGY.—Peritonitis may be diffuse or circumscribed, acute or chronic, but there are often transitions between these various forms. Diffuse peritonitis not infrequently begins with circumscribed changes, and may terminate in such. There are also transitions between acute and chronic peritonitis (subacute form). Acute exacerbations are often observed during the course of chronic peritonitis.

Peritonitis is rarely primary. This variety includes rheumatic and traumatic cases.

Every physician has met with cases in which no other cause could be discovered beyond a wetting while the body was heated, or perhaps lying upon the damp ground. In our opinion, a cold probably acts by favoring the development of certain low vegetable organisms.

Cold exercises an undeniable influence when the abdominal organs are in a congested condition, for example, during the period of menstruation.

Among the injuries which may produce peritonitis are a blow or fall on the abdomen, stab-wounds, explorations of the œsophagus in which the sound has passed into the peritoneal cavity.

Secondary peritonitis is conveyed most frequently from adjacent organs.

This category includes gastric ulcer or cancer, more rarely phlegmonous gastritis; duodenal ulcers, typhoid ulcers, tubercular ulcers of the intestines, dysentery, cancer of the intestine, coprostasis, typhlitis, invagination and volvulus; hepatic abscess, echinococcus, impaction of gall-stones; splenic abscess, infarctions, and echinococci; pancreatic abscess and calculi; renal abscess, paranephritis, perinephritis, pyelitis, impacted renal calculi; inflammation of the ovaries, tubes, uterus, or vagina; inflammation of the mesenteric and retroperitoneal glands, suppurating inguinal buboes, more rarely pleurisy and pericarditis. In such cases, the inflammation-producers are evidently carried to the peritoneum through the lymphatics.

In other cases peritonitis is the result of ulcerative processes in an abdominal viscus, extending to the peritoneum and producing inflammation directly. In some cases the ulceration leads to perforation of the organ, and various substances then enter the peritoneal cavity. This category includes almost all the causes mentioned above; in addition, rupture of abdominal aneurisms or abscesses into the peritoneal

cavity, extension of suppurative pleurisy or pericarditis, etc., etc. Indeed, it is hardly possible to mention in detail all the various lesions which may act in the manner referred to.

Peritonitis may also be associated with infectious diseases.

It is observed after scarlatina, diphtheria, small-pox, erysipelas, measles, and varicella. Bednar claims to have seen it a number of times after vaccination. It has also been claimed that peritonitis is intimately associated with acute articular rheumatism. I recently observed a case of acute articular rheumatism in which, at the beginning of the third week, undoubted signs of acute peritonitis developed, while the articular changes rapidly disappeared. At the end of ten days the symptoms of peritonitis subsided and the joint affection reappeared. Some cases are associated with syphilis, particularly the hereditary form. Peritonitis often occurs in pyæmia, septicæmia, and puerperal fever.

Circulatory disturbances, for example, valvular lesions or the blood changes in Bright's disease, morbus maculosus Werlhofii, or scurvy, sometimes give rise to peritonitis. Drunkards are also said to present a predisposition to the disease. It is sometimes associated with other affections of the peritoneal cavity (cancer, tuberculosis, ascites).

Peritonitis occurs at every age, but most frequently from the fifteenth to the fortieth years.

II. ANATOMICAL CHANGES.—The anatomical changes are very similar to those observed in pleurisy and pericarditis. We distinguish a dry (fibrinous) and a fluid (exudative) peritonitis. The latter may be serous, purulent, putrid, or hæmorrhagic, but there are numerous transitions between these forms.

The disease always begins as dry peritonitis. The subserous vessels are distended, so that the loops of intestines are unusually reddened. Extravasations of blood are observed not infrequently.

The peritoneum soon becomes cloudy, on account of swelling and partial desquamation of the endothelium cells. It is gradually covered with a thin, filmy membrane, which may be removed with the knife. This membrane gradually becomes thicker, yellowish, croup-like, and causes abnormal adhesion between the abdominal viscera. It consists of fibrin in parallel fibres inclosing cells.

The anatomical changes may now terminate. In rare cases, recovery results in the status quo ante. Liquefaction of the fibrin and fatty degeneration of the cellular elements occur, and finally the entire inflammatory product is absorbed. In the majority of cases the inflammatory products undergo partial organization, and result in the formation of fibrous adhesions or thickenings of the serous membrane. These may interfere with the movements of the abdominal organs, give rise to strangulation of the intestines, etc. If the peritonitis is very extensive, the peritoneal cavity may be obliterated, the mesentery and omentum undergo thickening and retraction, and the entire intestine knotted together into a coherent mass.

In many cases fibrinous peritonitis becomes converted into the exudative variety. In comparatively rare cases the fluid exudation is yellowish, poor in cells, often slightly cloudy and mixed with flakes of fibrin (serous or sero-fibrinous peritonitis). If the abundance of cells in the exudation increases, purulent peritonitis is the result. The pus may amount to thirty or forty litres. The loops of intestines are often adherent to one another by masses of fibrin, and on separating them we find deep pockets filled with pus. Putrid peritonitis, which occurs generally in puerperal fever, and after pyæmic and septicæmic conditions, originates from

putrid decomposition of the purulent exudation. The latter becomes acid, its ammoniacal odor is irritating to the nose, and it may even produce a pricking sensation when it comes in contact with the hands. The smell of sulphuretted hydrogen becomes apparent at a later period. The fluid is a stinking, grayish-green or brownish mass, which contains few cellular elements but numerous schizomycetes. Putrid peritonitis will develop as such from the start if the inflammation is the result of perforation of the intestine and the entrance of fæces into the peritoneal cavity.

Purulent exudation may have a faecal odor even if there is no communication between the peritoneal cavity and the intestine. If the presence of fæces is suspected, the pus should be examined with the microscope for particles of food. In one case Concato found *sarcina ventriculi* in the peritonitic exudation; the inflammation had resulted from perforation of a duodenal ulcer.

Hæmorrhagic peritonitis, like the serous variety, usually runs a chronic course. It is observed as a complication of cancer and tuberculosis of the peritoneum, also in morbus maculosus Werlhofii and scurvy. It often gives rise to a slaty discoloration of the peritoneum, the extravasated blood being converted into pigment. A special form of the disease was described by Friedreich. It occurred in a woman suffering from valvular disease of the heart, and in whom tapping for ascites had been performed sixteen times in one and one-half years. Upon the peritoneum was found a tumor, consisting of several laminated membranes, and between which more or less changed blood was situated.

At the beginning of exudative peritonitis the exudation collects in the dependent parts, particularly in the pelvis. At a later period, the distribution of the fluid is often irregular, on account of the numerous adhesions between various viscera.

Spontaneous recovery in fluid peritonitis is exceptional, and is especially rare in the purulent form. Purulent peritonitis affords opportunity for the recognition of the various complications of the disease. In some cases, the pus, by its eroding qualities, produces losses of substance in the peritoneum. These losses of substance may increase in extent, and terminate in perforation of pus into the intestines, stomach, pelvis of the kidney, ureter, bladder, through the diaphragm into the air passages, through the abdominal walls. The pus sometimes ruptures into two adjacent loops of intestines. Perforation into the abdominal blood-vessels may give rise to fatal hemorrhage or to pyæmia. In other cases, collections of pus remain encapsulated for a long time, but may subsequently form the starting-point for fresh exacerbations. The pus may also undergo caseation or calcification. Cheesy pus may prove the source of tubercular infection.

In diffuse peritonitis, the features are sunken; at the autopsy, the nose is peaked the eyes sunken, the abdomen distended. If the peritonitis is the result of perforation of the intestine, an incision into the abdomen is followed by an escape of gas, which has the odor of fæces or sulphuretted hydrogen, and burns with a bluish flame. Some authors believe that a purulent exudation may develop gas without preceding perforation.

The loops of intestines are markedly distended, the mucous membrane and muscular coat are pale, thickened, and infiltrated with serum. The serous membrane is often loosely adherent to the other layers of the intestines, and is readily stripped from the latter. The abdominal muscles and superficial layers of the parenchymatous organs of the abdomen are pale and succulent. The diaphragm may be pushed upward to the second or third rib.

III. SYMPTOMS.—We will first give the history of *acute, idiopathic, diffuse peritonitis*. The disease may or may not be preceded by prodromata. These consist of chill, repeated chilly sensations, fever, and gastro-intestinal disturbances.

Among the manifest symptoms, pain occupies a prominent part. It is referred to a circumscribed region, usually around the umbilicus, or to the entire abdomen. The slightest touch produces the most violent pain, and the patients can hardly bear the pressure of the lightest covering. For days and weeks they assume dorsal decubitus, the thighs drawn upward, and the knees slightly flexed to relieve the tension of the abdominal walls. The patients particularly dread the movements of vomiting, coughing, defecation, and micturition. The pain is described as boring, burning, or lancinating; occasionally there are colicky exacerbations.

The abdomen is usually more or less distended, and its walls are thin, smooth, and shining.

A peritonitic friction murmur is sometimes felt over various parts of the abdomen. The murmur is sometimes heard with the stethoscope, though it may not be perceptible to the touch.

Fluid exudation may give rise to fluctuation and dulness, but excessive tension of the abdominal walls may give rise to general dulness, and abolish fluctuation. In less degrees of tension, a metallo-tympanitic note will be produced, except in those places at which fluid is in contact with the abdominal walls. Dulness will be produced in such localities. In our experience, the dulness does not change with the changed position of the body.

Vomiting occurs in almost every case of peritonitis. At first, the ingested food is vomited, later yellowish or greenish matters, in rare cases faeces. Towards the end of life, vomiting sometimes ceases, and very obstinate singultus takes its place. If the vomiting continues, some of the vomited substances may enter the air passages, and give rise to foreign-body pneumonia.

The bowels are usually constipated, though diarrhoea is not infrequent at the beginning of the disease. The urine is scanty, very acid, of high specific gravity, and not infrequently contains small quantities of albumin.

An increased amount of indican and phenol has been found in the urine in peritonitis. The patients sometimes complain of pain in micturition or of inability to pass urine (implication of the serous coat of the bladder).

The bodily temperature is almost always elevated (40° C. or more at night). The type of fever varies. The pulse is usually very rapid; the radial pulse is not very full, but possesses considerable tension.

The features are expressive of pain, the eyes have a glassy look, are sunken, and are surrounded by bluish-gray rings. The cheek bones and nose become more prominent. The patients often speak in a whisper in order to avoid, as much as possible, movement of the diaphragm and abdominal walls. If they have vomited a good deal, the voice sometimes becomes hoarse.

Consciousness is often retained to the last, more rarely delirium or convulsions occur towards the end of life. The subjective complaints of the patient usually refer to pain in the abdomen, dyspnoea, anxiety, and unquenchable thirst.

As a rule, the tongue has a grayish-white or brownish coating; in

cases of obstinate vomiting, it often remains clean, very red, often dry and fissured. In some individuals, there is a very disagreeable foetor ex ore.

The respirations are almost always accelerated as the result of the fever, abnormal elevation of the diaphragm and compression of the lungs, pain on movement of the diaphragm and abdominal walls. The respirations are purely costal, the upper part of the thorax taking an especially active part in breathing.

The unusual elevation of the diaphragm is recognized by the fact, that the lower border of the lung is found on percussion beneath the fourth or even the third rib. Dulness on percussion is often heard over the posterior surface of the thorax inferiorly on account of the compression of the lung.

The elevation of the diaphragm is also shown by the unusual position of the heart. The apex beat may be displaced into the third intercostal space and several centimetres to the outside of the line of the left nipple. The cardiac contractions are usually very distinct over several intercostal spaces, and the second (diastolic) pulmonary sound is intensified. If marked meteorism is present, the heart sounds occasionally assume a metallic timbre, and are audible over a considerable portion of the abdomen.

Acute diffuse peritonitis may prove fatal in a few days, almost in a few hours. Death sometimes occurs unexpectedly, as if from shock; in other cases the meteorism becomes so excessive as to produce death by suffocation. The fatal termination may occur with signs of collapse.

The disease sometimes lasts for weeks, and passes into a subacute or chronic stage. This may be associated with various complications. For example, perforation of pus may take place. If it ruptures through the abdominal walls, the skin becomes red, swollen, and œdematous, fluctuation becomes noticeable, the skin grows thin, and finally the pus makes its escape. The umbilicus not infrequently constitutes the point of exit of the pus. The pus sometimes travels for a certain distance beneath the abdominal walls before it makes its escape. Perforation into the thoracic and abdominal viscera will be followed by corresponding symptoms (expectoration, vomiting, micturition of pus, etc.).

In some cases the deposits of pus are partly absorbed, in others they remain unchanged, but excite occasional acute exacerbations, and finally, after the development of marantic symptoms, prove fatal. After caseation and bacillary infection of the pus, there is danger of the development of tuberculosis. Even if the disease runs a very favorable and rapid course, gastro-intestinal disturbances are often left over (irregularity of the bowels, abdominal pains, etc.).

Secondary acute diffuse peritonitis may present the same symptoms as the primary form; but the fever and subjective symptoms are sometimes so slight that the peritonitis may be entirely overlooked, unless a careful objective examination is made. Friction murmurs, abnormal dulness, and fluctuation constitute the chief, sometimes the sole symptoms.

A few special remarks should be made concerning puerperal and infantile peritonitis.

In a series of cases, *puerperal peritonitis* is a benign inflammation which extends from the inner surface of the uterus; in others, it is a pyæmic or septicæmic condition, the product of certain bacteria which have entered the peritoneum. The first symptoms usually develop three to five days after delivery. The pains are often very slight during the entire course of the disease, but the

meteorism is unusually severe; there is almost always severe diarrhoea (sometimes of a dysenteric character), and frequent chills. In the septic form of puerperal peritonitis, death generally takes place from the end of the first to the middle of the second week.

Peritonitis infantum may be a foetal peritonitis. In a number of cases the signs of acute or chronic peritonitis have been found in new-born children; they were sometimes associated with syphilis, sometimes no cause could be ascertained. *Peritonitis neonatorum* is, in a certain sense, a traumatic disease, because it originates from the umbilical wound. Not infrequently it belongs to the same category as puerperal peritonitis, and is transmitted from the mother, or occurs in maternity hospitals during the prevalence of puerperal fever. Finally, peritonitis occurs not infrequently at a later period of childhood. Chronic peritonitis, particularly that form which is the result of tuberculosis of the peritoneum, is relatively frequent in childhood.

Acute local peritonitis is sometimes the starting-point of diffuse peritonitis, sometimes it remains circumscribed. According to its location, it is known as perihepatitis, perisplenitis, perimetritis, etc. The chief symptoms are pain, a perceptible and audible friction murmur, at times abnormal dulness and fluctuation; fever is not always present. In addition, there are functional disturbances of the corresponding organs.

Perforation-peritonitis sometimes proves fatal in an extremely short period, sometimes it runs a more chronic course (when it is preceded by the formation of adhesions, so that perforation takes place into an encapsulated cavity).

If the perforation of an organ occurs suddenly, the patients generally complain of intolerable pain, and may exclaim that something has torn within the abdomen. Sometimes they become almost unconscious. The face becomes pale, the pulse almost imperceptible, the limbs are cold as ice and covered with clammy sweat. In the beginning, the abdominal walls are hard as a board, depressed, and extremely sensitive to the slightest touch. This condition may continue unchanged, and terminate fatally in a few hours.

As a rule, perforation is followed by the entrance of the contents of the stomach or intestines, of pus, blood, bile, etc., into the peritoneal cavity, where they rapidly give rise to acute diffuse peritonitis. Pain, tympanites, vomiting, abnormal dulness, and fluctuation make their appearance. Vomiting is usually present except when the stomach is perforated; in such cases this symptom is often absent, because the gastric contents readily pass through the abnormal opening. If large blood-vessels have been perforated, the signs of internal hemorrhage appear.

If the digestive tract is perforated, gas enters the peritoneal cavity in addition to the solid contents of the intestines, and thus gives rise to pneumoperitonitis. If the gas is unimpeded in its movements, it rises and pushes the liver and spleen away from the abdominal parietes, so that the hepatic and splenic dulness disappears. This symptom will remain absent, if the liver and spleen are bound down by old adhesions, or if the gas enters a previously encapsulated space. In the latter event a succussion sound is produced on shaking the patient, and the boundary between the area of dulness and of the tympanitic sound in the gas-containing district varies with the changes in the position of the patient, the area of dulness always being situated below. Schudnewsky noticed that a metallic respiratory murmur is sometimes heard in intestinal perforation. This is evidently the result of a change in the respiratory murmur by resonance in the tympanitic peritoneal cavity.

Circumscribed chronic peritonitis often develops in a latent manner,

and is hardly the object of clinical observation. This includes the tendinous thickenings of chronic perihepatitis, perisplenitis, etc. It sometimes gives rise to a friction murmur, at other times to functional disturbances of the affected organ.

Diffuse chronic peritonitis may develop as such from the beginning, or it follows an acute attack. If peritonitis complicates tuberculosis or cancer of the peritoneum, it is generally chronic, almost always of a hemorrhagic type. Chronic peritonitis also develops in scurvy and Bright's disease.

The chief symptom is the presence of fluid in the abdominal cavity. Pain may be absent or very slight, and the patients sometimes continue to work for a considerable time. But symptoms on the part of the digestive organs are hardly ever absent, and if the fluid exudation is purulent, febrile movement will be observed.

The duration of the disease is almost unlimited. Some cases are incurable on account of the primary disease (cancer, tuberculosis). Thickening and exudation sometimes remain noticeable for life. In other cases death occurs from marasmus, or from suffocation, as the result of excessive exudation.

IV. DIAGNOSIS.—The recognition of acute peritonitis is usually easy. It may possibly be mistaken for the following diseases: *a. gastralgia*: the pain is confined mainly to the gastric region, is sometimes diminished on pressure, the patients are nervous and pale; *b. colic*: the pain is generally diminished on pressure, it moves from one place to another, borborygmus and flatulence are usually present; *c. renal or biliary colic*: we should look for changes in the functions of the liver or kidneys (jaundice, hæmaturia), and the passage of calculi in the stools or urine; the pain is localized in the right hypochondrium or the loins; *d. rheumatism of the abdominal muscles*: the pain is superficial, and often moves from one place to another.

Chronic peritonitis must be distinguished from ascites (vide following section).

V. PROGNOSIS.—The prognosis is always grave. Perforation-peritonitis is especially dangerous, although a few cases of recovery have been observed. The prognosis should also be given with caution, since serious consequences may develop very late (stenosis or volvulus of the intestines).

VI. TREATMENT.—When the disease is the result of incarcerated hernia, coprostasis, and the like, the treatment must be causal.

In acute peritonitis the patient should be kept absolutely quiet; the diet may consist of milk, soup, diluted claret mixed with pieces of ice. Small pieces of ice may also be given to relieve thirst. A thin, light poultice should be applied to the abdomen, and opium administered for days, even weeks (at first gr. ss. every hour, later every two hours). When the pupils contract, the intervals between the doses may be increased. In obstinate constipation, an intestinal infusion of water may be made every three to four days.

Some authors apply an ice-bag or cold compresses instead of warm poultices, but the former are not tolerated by many patients.

In violent vomiting and singultus, subcutaneous injections of morphine may be made, or chloroform may be inhaled. If the tympanites is so marked as to threaten suffocation, the intestines should be punc-

tured with a fine trocar. But this manipulation is not devoid of danger, since it may be followed by an escape of the intestinal contents. Bamberger recommended the introduction of an œsophageal bougie far into the rectum, but this plan is rarely effectual. In several cases we have had good results from intestinal infusions of ice-water. High fever is best combated by antipyrin (gr. lxxv. by enema) or thallin, gr. iv. every two hours until the temperature is reduced.

Chronic peritonitis must be treated by absorbents, but too much should not be expected from them. In addition to nourishing diet, we should order iodine, iron, iodide of iron, local applications of iodine, warm poultices, occasionally repeated blisters. Encapsulated purulent exudations have been treated successfully in a number of cases by incision and drainage. Abdominal puncture may become necessary in chronic serous peritonitis.

2. *Dropsy of the Peritoneal Cavity. Ascites.*

I. ETIOLOGY.—Ascites is an accumulation of fluid transudation in the peritoneal cavity; it is often associated with œdema of other parts.

It must be remembered, however, that ascites cannot always be distinguished with certainty from peritonitis; moreover, there are sometimes transitions between the two processes.

Ascites is always the result of one or two factors, viz., abnormal increase of the blood pressure, or unusual permeability of the walls of the vessels.

The circulatory disturbances are sometimes local, sometimes they originate in the heart.

The chief local diseases are disturbances of the portal circulation, whether they are situated in the radicles of the portal vein, in the trunk itself, or in the intra-hepatic branches.

As we have already remarked in previous sections, ascites is a symptom of various hepatic diseases, and of compression of the portal vein and inferior vena cava from various causes.

Ascites may also be the result of a disease of the peritoneum itself (cancer, tuberculosis).

Among the general circulatory disturbances which may give rise to ascites are chronic diseases of the heart and respiratory apparatus which interfere with the complete emptying of the right heart, and thus cause stasis in the superior and inferior venæ cavæ. In such cases, the œdema appears not alone as ascites, but even earlier in the lower limbs, and later in the pleural and pericardial cavities.

Abnormal permeability of the vessels develops if the organism suffers severe losses, and the blood is thus impoverished in albuminoids (Bright's disease, protracted diarrhœa or suppuration, frequent losses of blood, exhausting diseases, etc.). Under such circumstances, ascites is associated with œdema in other localities. Not infrequently circulatory disturbances and changes in the blood-vessels act together in producing ascites.

Ascites may occur at any age, but most frequently from the 15th to 40th years. It has also been observed in the new-born, as the result of hepatic and splenic diseases, or severe illness of the mother during

pregnancy. The children often present various deformities, such as hare-lip, etc. Congenital ascites may be so abundant as to prove an obstacle to delivery.

II. ANATOMICAL CHANGES.—The abdominal cavity may contain ten to twenty litres, or more, of fluid. It generally has an amber-yellow color, but is sometimes almost as clear as water; if jaundice is present, it has a yellowish cast (green in reflected light). In cancer or tuberculosis of the peritoneum, and after puncture, the fluid sometimes presents a sanguinolent appearance; this may change to brown, from changes in the blood pigment. The fluid not infrequently contains flocculi and sometimes shining crystals (cholestearin). The flocculi are composed mainly of desquamated, swollen endothelium-cells of the peritoneum. The reaction is almost always alkaline, more rarely neutral, most rarely acid. The specific gravity varies from 1.004 to 1.014.

The fluid is poor in microscopical constituents. It contains round cells, a few red blood-globules, desquamated endothelium-cells, granular masses, and fibrin.

The transudation contains constituents of the blood plasma. Serum albumin, serum globulin, fat, urea, uric acid, xanthin, kreatin, kreatinin, leucin, and sugar have been found in it. Under certain circumstances, it contains large amounts of substances which are ordinarily present in minute traces. Thus, in a patient who had passed no urine in three days, Daremberg found 6 gm. urea in 1,000 ccm. ascitic fluid.

Hoffmann found that the amount of serum albumin and serum globulin varies greatly in different cases, and that both are present in smaller quantities than in the serum of the blood, but that their relative proportion is the same as in the serum.

It was found that the presence of fifteen to twenty parts of albumin per thousand of fluid indicated a transudation; in exudations, there were more than forty parts per thousand. The amount of albumin is especially small in ascites following waxy degeneration of glandular organs. According to Hoffmann, diseases of the portal vein and peritoneum may be excluded if the fluid contains less than ten parts per thousand of albumin.

To make a rapid estimate of the amount of albumin in the fluid, Reuss recommended the following formula: $E = \frac{2}{3}(S - 1,000) - 2.8$, in which E = percentage of albumin, and S = the specific gravity of the fluid.

If the ascites is complicated by peritonitic changes, the amount of albumin increases at once.

Fatty and chylous ascites constitute special forms of the disease.

Fatty ascites has been observed in cancer and tuberculosis of the peritoneum and in chronic peritonitis. The fluid has a milky appearance, and a layer of cream forms after standing. This is the result of admixture with desquamated tumor-cells, which are fatty and partly dissolved. The fluid is alkaline; its specific gravity varies from 1.012 to 1.023. Ormerod furnishes the following analysis:

Specific gravity,	1012.05
Water,	947.73
Solid substances,	52.27
Albumin,	17.26
Casein-like masses,	2.39
Fats,	19.93
Sodium chloride,	6.51
Biliary constituents,	} 6.18
Sugar,	
Phosphoric acid,	
Lime and unascertainable substances,	

Chylous ascites is an accumulation of more or less pure chyle in the abdominal cavity. Rupture of the chyle ducts has not been observed in all cases. Stern gives the following analysis:

Specific gravity,	1023.	
Water,	89.88	per cent.
Solid constituents,	10.12	" "
Albumin,	5.634	" "
Fats,	3.300	" "
Sugar,	0.032	" "
Ash,	0.310	" "
Peptone in traces,		

In very extensive ascites of long standing, the abdominal organs undergo compression atrophy, and are often unusually pale. This is also true of the abdominal muscles. The peritoneum not infrequently presents opacities and thickenings, sometimes granular prominences, composed of proliferated endothelium-cells.

Œdematous accumulations between the layers of the omentum are often observed in children.

III. SYMPTOMS.—We may regard 500–1,000 ccm. fluid as the smallest amount which can be demonstrated objectively in the abdominal cavity. As this amount of fluid will settle in the pelvis, its recognition requires special methods of examination. Bamberger proposed that the patient should lie with the sacrum as high as possible; the fluid will then flow into the region of the loins, and there give rise to dulness. Or, if the patient assumes the knee-elbow position, the fluid will flow upon the anterior abdominal wall, and produce dulness around the umbilicus.

If the amount of fluid is large, a great number of subjective and objective changes are produced.

The patients generally complain of a feeling of unusual tension and fullness in the abdomen. Dyspnœa is usually noticeable on account of the interference with the movements of the diaphragm and compressions of the lungs. Anorexia and constipation are commonly present; singultus and vomiting are also frequent symptoms.

Changes in the size and shape of the abdomen are evident on objective examination. While the belly is prominent anteriorly, the chest is inclined backwards. The shape of the abdomen often changes with the position of the body. In the erect position, the lower half of the belly projects anteriorly to a marked degree, in the recumbent posture the anterior surface of the abdomen is more flattened, the sides are more prominent.

The abdominal integument is usually very pale, smooth and shining; it is tense, and often unusually thin. In very extensive ascites the cutis ruptures in places, which appear as rosy-red or bluish-red stripes. They appear first and in greatest numbers in the lower lateral portions of the abdomen. They usually persist for life, and finally assume a cicatricial, whitish appearance.

The abdominal integument is sometimes œdematous, but, as a rule, not unless anasarca of the lower limbs and external genitals is also present. If the œdema of the skin becomes excessive, it may terminate in erythematous and erysipelatous changes, oozing of fluid, fissures and gangrenous processes.

The subcutaneous abdominal veins are often visible as sinuous, sometimes very dilated strands. They usually run upwards in two main

trunks from the middle of Poupart's ligament towards the umbilicus and communicate with two veins which run to the thorax. This dilatation of the epigastric veins may be the result of compression of the inferior vena cava by the ascitic fluid. Disturbances in the portal circulation may produce the so-called caput medusæ.

The umbilicus is often effaced; if the ascites is very abundant, the navel often protrudes like a hernia. In one of my cases there was marked separation of the recti muscles and the development of a large umbilical hernia.

Palpation is very important in the examination of ascites. If the index or middle finger of the right hand gives a quick stroke to one side of the abdomen, while the palm of the left hand is applied to the other side, the wave-like movement of the fluid will be felt as fluctuation. These movements may also be seen not infrequently beneath the abdominal walls. Very extensive ascites and too great tension of the abdomen may diminish or even abolish fluctuation. In other cases, the waves are so small and numerous as to resemble hydatid tremor.

On percussion, dulness is found wherever the fluid is in contact with the abdominal walls. A characteristic feature is the change in dulness according to the position of the body. In dorsal decubitus, the lateral and lower parts of the abdomen are dull; the anterior upper parts are tympanitic on percussion. This is owing to the fact that the intestines, which are filled with gas, float on the surface of the fluid. In lateral decubitus, the free, upper side of the abdomen is tympanitic on percussion—in short, the intestines always float upon the surface of the fluid. The upper boundary of dulness forms a wavy line, owing to the variable degree of entrance of the fluid between the loops of intestines.

These changes on percussion will not be produced if the amount of fluid is excessive, if the mesentery is retracted as the result of previous inflammation, or if the loops of intestines are adherent to one another.

Auscultatory symptoms may be entirely absent. In some cases, sudden movements of the patient will produce a peculiar swash in the fluid.

The heart and lungs are pushed upwards, the lower lobes of the lungs being often compressed. The patients often have no rest unless they assume the sitting posture (orthopnœa). Signs of cyanosis are generally present. Diuresis is almost always diminished.

The disease may last many months, even years. Death may result from the primary disease or from suffocation (vigorous compression of the thoracic organs).

IV. DIAGNOSIS.—The diagnosis depends mainly on two symptoms, viz., fluctuation, and the changes in percussion on changing the position of the body. The fluid is distinguished from peritonitic exudation by the etiology, and the absence of fever and pain. In certain forms of chronic peritonitis, the diagnosis may be doubtful.

The percentage of albumin in the fluid (obtained by puncture) may be employed in the differential diagnosis. According to Reuss, twenty parts albumin per thousand indicates a transudation. The fluid cannot be positively regarded as inflammatory, however, unless it contains forty parts albumin per thousand, since there are certain stages of transition between transudations and exudations. The specific gravity is also important in diagnosis. Hoffmann distinguished three forms of ascites: *a.* cachectic ascites, for example, in chronic nephritis, in which the specific gravity is less than 1.010; *b.* inflammatory ascites, in which the

specific gravity is above 1.014; and *c.* stasis ascites, in which the specific gravity varies between the figures mentioned above.

Ascites may be mistaken for ovarian cysts even by experienced gynecologists. The following points should be taken into consideration in differential diagnosis:

a. In dorsal decubitus, the abdomen is flattened anteriorly in ascites, sharply convex in ovarian cysts.

b. In ascites, the umbilicus is effaced or projecting; in ovarian cyst it is simply pushed upwards.

c. The enlargement of the abdomen is uniform in ascites; in ovarian cysts there is often a prominence in certain parts.

d. In ascites, the percussion sound in dorsal decubitus is tympanitic anteriorly, dull laterally; in ovarian cysts the reverse holds good.

e. The boundary of dullness is wavy in ascites, straight in ovarian cysts.

f. Changes in percussion on changing the position of the body remain absent in ovarian cysts.

g. The feeling of fluctuation extends beyond the area of dullness in ascites, is limited to this area in ovarian cysts.

h. In ascites, the uterus is depressed; in ovarian cysts it is generally elevated or in a lateral position.

i. The fluid has a less specific gravity in ascites (1.010-1.014) than in ovarian cysts (1.018-1.024).

j. In ascites, the fluid is usually clear and thin; in ovarian cysts it is often cloudy and thicker.

k. The ascitic fluid contains endothelium cells, the fluid of ovarian cysts cylindrical cells.

After the existence of ascites is recognized, we should endeavor to ascertain its causes. If ascites exists alone or develops first, it may be attributed to disease of the liver, portal vein, peritoneum, or an abdominal organ; the differentiation between all these conditions will depend upon the changes in the individual organs and upon the functional disturbances. Ascites may interfere with the examination of the abdominal viscera, and our opinion must then be reserved until the fluid is removed by puncture.

V. PROGNOSIS.—This depends upon the primary disease. The ascites *per se* may be a source of danger and give rise to suffocation. According to Méhu the prognosis is so much more unfavorable the lower the specific gravity of the fluid.

VI. TREATMENT.—In the first place, causal treatment must be adopted, and this varies according to the character of the primary disease. Drastics, diuretics, and diaphoretics are the chief remedies which must be relied upon.

The ascites itself is often treated successfully by local measures. The most prompt in its action is puncture of the abdomen.

This may be done with an ordinary trocar, which has been thoroughly heated and then cleaned with a five-per-cent solution of carbolic acid. The patient should be seated in a comfortable arm chair. The incision is made as low as possible, and the fluid removed at intervals in order to prevent cerebral anæmia from sudden removal of the pressure on the abdominal vessels. To facilitate the discharge of the fluid, the abdomen is surrounded by a bandage which is gradually drawn tighter and tighter. After the tapping is completed, two long Carlsbad needles are passed at right angles through the edges of the incision, and the latter then closed by a thread drawn around the needles. A bandage is then drawn firmly around the abdomen to prevent the rapid reaccumulation of the fluid. Liebermeister has recently recommended that a glass funnel be connected with a rubber tube, the whole filled with a five-per-cent solution of sodium salicylicum, and the funnel then rapidly applied over the incision and fastened with adhesive plaster. This apparatus acts as a siphon and facilitates the continued discharge of the fluid.

Tapping has produced fatal hemorrhage in very rare cases as the result of injury of large vessels. Inflammation of the peritoneum has also been observed, but probably as the result of the employment of dirty instruments. The entrance of air into the abdomen need not be feared, since the intestine is situated directly behind the opening of the canula. But if we dread the occurrence of this accident, tapping may be performed with the aid of a condom in the manner described in Vol. I., page 367. If the intestines occlude the opening of the canula at the start, they must be pushed away by introducing a thoroughly cleaned and carbolyzed bougie into the canula.

Tapping must sometimes be repeated very often; in Lécane's case, the operation was performed eight hundred and eighty-six times.

Some writers have obtained favorable results from faradization of the abdominal walls. A strong current is employed, one electrode is placed on the loins, the other over the motor points of the abdominal muscles, which are brought into contraction fifty to one hundred times at each sitting. The applications produced a rapid increase of diuresis and then disappearance of the ascites.

Mackenzie has successfully treated ascites by the application of flannel bandages.

Spontaneous recovery has occurred in very rare cases from perforation of the fluid through the umbilicus, scrotum, intestine, or, as in Ringland's case, through the Fallopian tubes.

3. *Cancer of the Peritoneum.*

Cancer of the peritoneum is rarely primary. It is generally secondary to cancer of the stomach, intestines, liver, retroperitoneal glands, etc., and develops either as a metastasis or is propagated directly from the organs mentioned. The tumors may attain very considerable dimensions (one hundred and fourteen pounds in one case), and form round prominences or diffuse infiltrations. In rare cases, which generally run an acute course, it appears in the form of numerous nodules like miliary tubercles. It is often associated with peritonitis or ascites, which is frequently hemorrhagic in character. Fatty ascites is also found in this affection (vide page 238). The cancer may ulcerate and give rise to dangerous hemorrhage, or it may rupture into the abdominal organs.

The diagnosis depends on the demonstration of tumors which cannot be attributed to any of the abdominal viscera, and the symptoms of peritonitis or ascites, associated with cancer in other organs. The treatment is purely symptomatic.

4. *Parasites of the Peritoneum.*

a. Echinococci have been observed upon the peritoneum in a few cases; they were sometimes associated with echinococci in other organs. A fluctuating tumor is found, and this not infrequently presents hydatid tremor. Enlargement of the hydatids produces signs of compression and may prove fatal by interfering with the movements of the diaphragm. If the echinococcus is isolated, the treatment is surgical; if not, it is symptomatic.

b. *Cysticercus cellulosæ* and *pentastomum denticulatum* have been observed in rare cases. In one case Winkel found *filaria sanguinis* in a buttermilk-like fluid which was removed from the abdomen.

c. Parasites, especially ascarides, not infrequently enter the peritoneal cavity in perforation-peritonitis.

SECTION IV.

DISEASES OF THE URINARY AND SEXUAL APPARATUS.

PART I.

SYMPTOMATICALLY IMPORTANT CHANGES IN THE URINE.

1. *Albuminuria.*

1. The presence of dissolved albumin in the urine constitutes albuminuria. This condition has been divided into three forms, true, false and mixed albuminuria. In true albuminuria, the albumin has entered the urine within the kidneys; in false albuminuria, it has entered the urine accidentally along the urinary passages; mixed albuminuria is a combination of both the other forms.

True albuminuria is again subdivided into the renal and hematogenous forms. The former is the result of anatomical changes in the kidneys, the latter of changes in the blood.

In some cases albuminuria lasts for months, even years (permanent), in others it is a temporary phenomenon, perhaps continuing only for several hours (transitory).

2. As a rule, we can readily distinguish between true and false albuminuria. In the latter, the amount of albumin is usually slight and corresponds to the amount of pus or blood which is mixed with the urine.

3. The most important of the various albumins in true albuminuria is serumalbumin. In many cases serumglobulin (paraglobulin) is also found in the urine, and Estelle and Werner even observed serumglobulinuria to the exclusion of albuminuria. Peptone may also be present in the urine. In peptonuria the urine may remain perfectly clear on the application of the ordinary tests for albumin (boiling and nitric acid). Gerhardt applied the term latent albuminuria to such cases. He observed pure peptonuria in febrile conditions, and in all inflammatory diseases as soon as the products of inflammation undergo absorption (pus-corpuscles contain peptone). Maixner observed peptonuria in gastro-intestinal affections; Jaksch found it in scurvy. Fischl noticed it during the puerperal condition, in the majority of cases, for several days after delivery. Hemialbumose (propeptone) has also been found in the urine. Senator and Grigoriantz showed that propeptonuria is by no

means so very rare. They found it under very different circumstances, often in combination with ordinary albuminuria and peptonuria. It is also said that paralbumin and metalbumin have been found in the urine.

4. The following are the tests employed to determine the presence of albumin (serumalbumin) in the urine.

a. The best known one is the heat and nitric acid test. A test-tube is filled one-fifth full with urine, it is then heated to boiling, and then nitric acid is added (about one-tenth the volume of urine). If the urine contains a small amount of albumin, it will become diffusely cloudy; if a larger quantity is present, more or less flakes will be deposited. If the amount of albumin is very considerable, the urine may be converted into a firm mass.

Feebly acid or neutral urine often becomes cloudy or flocculent on heating on account of the precipitation of phosphates. This cloudiness disappears on the addition of nitric acid.

In individuals who have taken balsams, for example, copaiba or turpentine, the urine may become cloudy on the addition of nitric acid, although it contains no albumin. That this is a resinous precipitate is shown by the fact that it redissolves on the addition of alcohol.

b. Heller's test. A test-tube is filled about one-fourth full with pure nitric acid, and filtered urine is then poured in cautiously along the side of the tube so that the urine forms a layer above the nitric acid. A white ring of albumin forms at the point of contact of the two fluids.

A brown ring of urinary pigment is sometimes found a little above the lowermost layer of urine; its lower border fades away very gradually.

If the urine contains an abundance of urates, a ring is sometimes formed similar to that produced by the albumin, but the former is situated lower than the latter, and in addition it disappears on heating.

c. Heat and acetic acid. This test is carried out in the same manner as the heat and nitric acid test. If too much acetic acid is added, a soluble acid albuminate is formed, so that the precipitation of albumin does not take place, or a precipitate which has formed again dissolves. It is best to add 2 drops acetic acid to 15 ccm. urine.

Acetic acid also precipitates mucin, but this is undissolved by an excess of acid.

d. Picric acid (Galippe). If urine which contains albumin is filtered and an excess of concentrated picric acid is added, a flocculent precipitate will be produced or, if the amount of albumin is very small, a simple cloudiness. Cooke and Watkins claim that picric acid will produce a precipitate in non-albuminous urine which contains quinine or potash salts, but this is not confirmed by my own experience. Peptones can also be recognized with the aid of picric acid; they are also precipitated by the acid, but, unlike albumin, are redissolved on boiling or on the addition of nitric acid.

e. Metaphosphoric acid (Hindelang). A little metaphosphoric acid is dissolved in distilled water and then added to filtered urine. If the latter contains albumin, a white cloudiness will form. This test will also detect peptones in the same way as does picric acid.

There are numerous other tests for albumin, but these are sufficient for all practical purposes.

The tests mentioned will not distinguish serumalbumin from paraglobulin. In order to do this, magnesium sulphate is added to the urine until the latter is saturated and no longer dissolves the salt. The precipitate, which then forms, consists of paraglobulin. If the urine is then filtered, the paraglobulin remains on the filtering paper, and the serumalbumin passes through. The latter can then be tested in the usual way.

Hemialbumose (propeptone) is characterized by the fact that the urine remains clear on boiling; it becomes cloudy, when cold, on the addition of acetic or nitric acid, and again clears on warming.

Peptone is not precipitated on boiling or on the addition of nitric or acetic acid, but it is precipitated by alcohol, metaphosphoric and picric acids. Pure peptonuria may be assumed to be present if picric and metaphosphoric acids produce a precipitate, but the other tests for albumin give negative results. When peptone is precipitated by picric acid, it redissolves on boiling, or the addition of nitric acid.

The amount of albumin in the urine varies from a trace to 30 gm. in the twenty-four hours. As a matter of course, large losses of albumin impair the nutrition, but this factor should not be overestimated.

An approximate notion of the amount of albumin in the urine may be obtained if a graduated test-tube is filled to a certain point with urine, to another certain point with nitric acid, and then, after boiling, the tube allowed to stand for twenty-four hours. The height of the precipitate will then furnish an approximate idea of the amount of albumin excreted.

5. Albuminuria sometimes occurs in healthy individuals (physiological albuminuria). Chateaubourg found it particularly in the urine which was passed during the morning. In others it occurs after a long walk or depressing emotions. Some individuals have transitory albuminuria after eating eggs, especially raw eggs, or after any hearty meal. Bence Jones noticed albuminuria following cold baths. According to Chateaubourg, it may also occur after sexual excitement. It occurs occasionally after profuse perspiration. It is often found in new-born infants during the first few days of life.

We will now give a brief sketch of the various morbid factors which may give rise to albuminuria:

a. Febrile albuminuria is a frequent symptom. It is observed especially in febrile infectious diseases, when the temperature has ranged above 40° C. for some time. Marckwald's observations render it probable that in some cases the infectious process, irrespective of the fever, is the cause of albuminuria.

b. Nervous albuminuria is that form which follows severe nervous diseases. It has been observed after cerebral hemorrhage, the apoplectic attacks of general paralysis, epileptic attacks, delirium tremens, meningitis, etc.

c. Anæmia, cachexia, and sudden, profuse losses of blood are not infrequently the cause of albuminuria.

d. The symptom possesses intimate relations to cutaneous changes. According to Capitan, albuminuria sometimes occurs after faradization of the skin; likewise after inunction with irritating substances (iodine, tar, etc.)

e. Toxic albuminuria is the result of the introduction of poisons into the body. They act directly on the kidneys, or indirectly by producing

disturbances of innervation. Among these poisons are mineral acids, phosphorus, cantharides, morphine, chloroform, etc.

f. Circulatory disturbances may be the cause of albuminuria, if they produce stasis in the inferior vena cava and thus in the renal veins; thrombosis of these veins acts in a similar manner.

g. Renal albuminuria is the term applied to those cases in which renal lesions produce albuminuria. But this is not a necessary consequence of all renal lesions, particularly of circumscribed ones.

h. Albuminuria is sometimes the result of occlusion of the urinary passages; for example, of occlusion of the ureter by a calculus or tumor.

6. The Malpighian bodies are generally regarded as the site at which the albumin is excreted. Some writers believe that an active part is also taken by the convoluted tubes.

According to Heidenhain, albuminuria is produced by every condition which interferes with the integrity and function of the epithelium of the Malpighian bodies, since it is their function to retain the albuminoids in the blood under normal conditions. Slight changes in circulation, especially slowness of the current, suffice to interfere with the function of the epithelium cells, and this disturbance becomes more marked in the gross anatomical changes of nephritis. In all probability the function of these cells is also affected by the constitution of the blood, and thus explains the albuminuria of anæmia and cachexia.

7. The prognosis and treatment naturally depend on the primary disease. Fuchsin (gr. iss.-ivss. in powder every day) has been recommended as a, to a certain extent, specific remedy, but we have obtained no good results from this drug.

In all forms of albuminuria, the chief stress should be laid upon the dietetic treatment. The patients should always wear thin woollen underclothing; warm baths (28–30° R.) should be taken twice a week; cold baths should be avoided. In winter the patients should visit a warm climate. A long stay in bed also acts very favorably in many cases.

Strong coffee, tea, alcoholics, eggs, and spiced articles should be avoided; a mild claret may be taken. An exclusive meat diet increases albuminuria; fish is the best form of food in this category. The patient should be advised to partake abundantly of fats and milk. The meals should be light but frequent.

Thirst may be relieved by alkaline-muriatic or alkaline-acidulous waters (Ems, Selters, Vichy, Bilin, etc.).

2. *Hæmaturia.*

(*Mictus cruentus.*)

I. ETIOLOGY.—*Hæmaturia* is that condition of the urine in which it contains so many red blood-globules as to present a characteristic color. In hæmoglobinuria the urine contains only the coloring matter of the blood.

Hæmaturia is merely a symptom of various diseases of the kidney, pelvis of the kidney, ureters, bladder, and urethra.

a. Among renal diseases hæmaturia is observed very often as the result of injury to the organs. In rarer cases it is the result of a cold, as in a case reported by Socoloff, in which hæmaturia occurred whenever

the patient was exposed to the wet. The entire process in this case created the impression of a vaso-motor disturbance.

In some cases renal hæmaturia is the result of poisoning with cantharides, turpentine, quinine, salicylic acid.

It is sometimes the result of diseases of the renal vessels (embolism, thrombosis, aneurism, stasis), but occurs very rarely in waxy degeneration of the kidneys. It is a frequent symptom of inflammations and neoplasms of the renal parenchyma. In acute nephritis, hæmaturia is an almost constant symptom; in chronic nephritis it appears during acute exacerbations. It has been described a number of times in echinococcus of the kidneys.

Hæmaturia also occurs occasionally in certain infectious diseases, rarely in some forms of syphilis, with relative frequency in intermittent fever. It has also been observed in small-pox, measles, scarlatina, typhoid, typhus and relapsing fevers, etc.; but the blood is then derived, as a rule, from the mucous membrane of the renal pelvis. It would also seem as if febrile diseases *per se* may give rise to such a degree of renal congestion as to result in hæmaturia. This symptom has also been observed in certain blood diseases (scurvy, morbus maculosus Werlhofii, purpura, urticaria, and hæmophilia).

b. Hæmaturia from diseases of the pelvis and ureters is produced most frequently by renal calculi and tuberculosis. Parasites are an occasional cause; for example, distomum hæmatobium and filaria. In rare cases this form of hæmaturia is the result of rupture of abscesses of the neighboring parts into the urinary passages.

c. Among diseases of the bladder, calculi and cancer give rise most frequently to hæmaturia. The blood is sometimes derived from dilated veins of the vesical mucous membrane, more rarely from violent cystitis, still more rarely from ulcerations. In tropical regions vesical hemorrhage may be produced by distomum hæmatobium.

d. Urethral hemorrhages are generally the result of injury (catheterization, impaction of calculi, etc.), more rarely they appear during the course of gonorrhœa.

In a certain proportion of cases of hæmaturia, no cause can be ascertained.

It is more frequent in men of middle age than in females.

II. SYMPTOMS.—If a small amount of blood is diffused uniformly through the urine, the latter often presents a pale rosy color. The more blood it contains, and the more rapidly it is discharged, the more distinct becomes its blood-red color. If micturition is delayed, the transformation of the blood pigment imparts a reddish-brown, blackish-brown, or blackish-green color to the urine. If the blood is mingled uniformly with the urine, the latter becomes dichroitic, *i. e.*, it is greenish in transmitted light.

If the blood is derived from the urethra, the color of the urine may be unchanged, and bloody clots will be found at the bottom of the vessel.

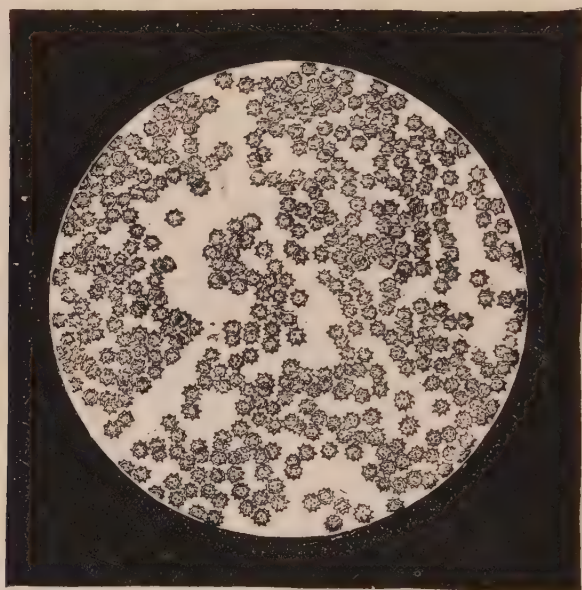
The urine is at first opaque and cloudy, but after standing it deposits a brownish-red sediment, while the upper layers become clear. The reaction generally remains acid, except in a few cases of vesical hemorrhage, in which the hemorrhage may be so great that the urine becomes neutral or alkaline. Of course, this does not hold good with regard to those cases in which a long-standing vesical disease leads to ammoniacal

decomposition of the urine. In such instances the urine remains alkaline from the presence of volatile alkalis.

In the majority of cases, the sediment forms a granular, crumbly red or brownish-red layer. Blood clots are observed much less frequently. This is especially true when the hæmaturia is the result of diseases of the renal parenchyma, although clots are sometimes present even under such circumstances. The clots are observed more frequently in hemorrhages from the pelvis of the kidneys or the ureters.

In doubtful cases, red blood-globules are readily recognized, as a rule, under the microscope. The shape of the blood-globules is often unchanged, but they are usually separate, rarely nummular. In some cases they lose their biconcave shape and become spherical; at the same

FIG. 64.



Mulberry-shaped red blood-globules in the urinary sediment in hæmaturia.

time they are smaller and more deeply colored. Under such circumstances the blood-globules are often very unequal in size. If the urine is very concentrated, the mulberry shape is not infrequent (vide Fig. 64).

The red blood-globules sometimes have a peculiar brownish-red color, sometimes are entirely colorless from loss of pigment. They may even be indistinguishable except on the addition of a solution of iodine and potassium iodide (iodi puri, gr. viiss.; potass. iodid., gr. lxxv., aq. destil., $\bar{3}$ iiiss.), which colors them yellow.

Friedreich noticed that the red blood-globules sometimes manifest amœboid movements. These movements may continue for twelve hours and, according to Friedreich, are characteristic of renal hæmaturia (Fig. 65). The experiments of Koelliker, Preyer and Kneulinger have shown that this phenomenon may be produced by the action of solutions of urea on the blood-globules.

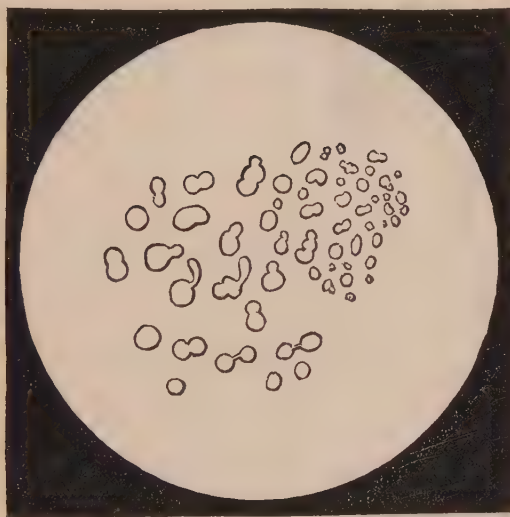
In renal hæmaturia any casts which may be present are often lined with blood-globules, and the latter are sometimes aggregated into cylindrical structures (blood casts). Hæmatoidin crystals are sometimes visible.

The spectroscopic examination for blood in hæmaturia is rarely employed in medical practice, but it becomes necessary if we wish to determine the presence of different blood pigments in the urine. The following are the principal data in this method of examination:

(a) Oxyhæmoglobin gives two absorption bands in yellow and green between the Fraunhofer lines D and E, the one nearer to D being the more sharply defined (vide Fig. 66, I.). Oxyhæmoglobin rarely occurs alone in hæmaturia.

(b) If oxyhæmoglobin is reduced by shaking it with a few drops of ammonium sulphide, a spectrum is produced in which the two absorption bands coalesce into one (Fig. 66, II.). The single band fills up the interval between the two bands

FIG. 65.



Red blood-globules in amœboid movement, in renal hæmaturia. After Friedreich.

of oxyhæmoglobin. Reduced hæmoglobin is found not infrequently in hæmaturia if the urine is decomposed.

(c) Methæmoglobin is observed almost constantly in hæmaturia. In a single molecule it contains less oxygen than does oxyhæmoglobin, and has an absorption band in red between C and D (Fig. 66, III.).

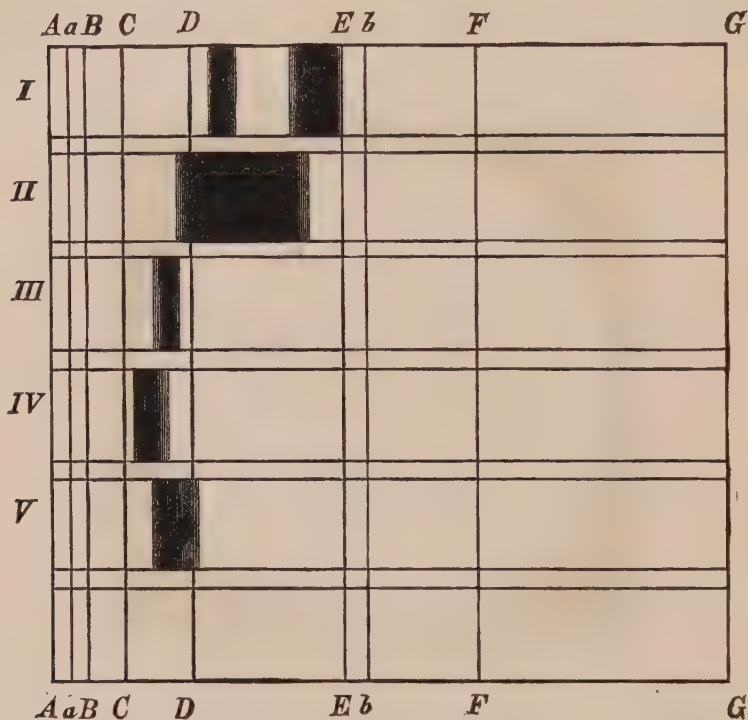
(d) Hæmatin does not occur in the urine, according to Hoppe-Seyler, but this has been denied by others. In an acid solution it has a characteristic absorption band in red near C (Fig. 66, IV.), in an alkaline solution the band is nearer D (Fig. 66, V.).

Heller's test for blood is very useful in practice, because it is very simple and exact. It is made as follows: A little urine is poured into a test-tube, about one-third the quantity of a solution of potash (1 : 3) is added and then boiled. The earthy phosphates are precipitated in large flakes, which have a light gray color if the urine contains no blood. If blood is present, the blood pigment is decomposed and free hæmatin is

deposited with the flakes, so that they assume a brownish-red or ruby-red color. These flakes are also dichroitic, *i. e.*, they are red in reflected light, green in transmitted light. If the urine has undergone ammoniacal decomposition, an equal volume of normal acid urine should first be added before the test is applied.

Teichmann's test is made in the following manner: Some urinary sediment is placed on a slide, a very small grain of salt added, three to five drops of pure acetic acid allowed to flow in under the cover glass, and the slide then heated over a spirit lamp until bubbles form. When it grows cool, we will find, with the aid of the microscope, crystals of hæmin (rhombic

FIG. 66.



Absorption spectrum of the various modifications of blood-pigment.

I., oxyhæmoglobin; II., reduced hæmoglobin; III., methæmoglobin; IV., hæmatin in acid solution; V., hæmatin in alkaline solution.

tablets or rods) sometimes irregularly formed granules of hæmin (Fig. 67).

Very profuse and protracted hæmaturia may give rise to dangerous signs of anæmia. Hemorrhages in the urinary passages sometimes form the starting-point of calculi.

III. DIAGNOSIS.—The recognition of hæmaturia is not difficult if we take into consideration the tests described in the previous section.

In renal hæmaturia, the blood is mingled uniformly with the urine, the latter generally contains more albumin than is accounted for by the blood, and the sediment generally contains casts.

In hemorrhages from the pelvis of the kidneys and ureters, the appearance of cylindrical clots is important; the etiology must also be taken into consideration.

Hemorrhages from the bladder are often associated with pain in the region of the bladder, dysuria, and other disturbances of micturition. Large clots in the urine, its alkaline reaction immediately after passage or the appearance of cast-off shreds of vesical tumors, favor the diagnosis of hemorrhage from the bladder.

In urethral hemorrhage, blood can generally be squeezed from the meatus by pressure on the urethra, and small amounts of blood are passed at the beginning of micturition. If the hemorrhage originates at the neck of the bladder, a few drops of blood will be discharged, attended with violent pain, at the end of micturition.

IV. PROGNOSIS.—This depends upon the primary disease, but it

FIG. 67.



Hæmin crystals, obtained by Teichmann's test. Enlarged 300 times.

should not be forgotten that very profuse hæmaturia may be a direct source of danger.

V. TREATMENT.—In the majority of cases, we need only treat the primary disease. If the loss of blood is very considerable and protracted, we should order absolute rest in bed, an ice-bag over the site of hemorrhage, and subcutaneous injections of ergotinum Bombelon (one syringe-ful mixed with equal parts of water t. i. d.). Various styptics may also be employed (vide page 56). Caspari obtained good results from phosphate of lime, Upshur from tinct. guaiaci (70 drops t. i. d.) In vesical hemorrhage we may make injections of ice-water, nitrate of silver (gr. $\frac{3}{4}$ -viiss. : $\frac{3}{4}$ iiiss.), or liq. ferri sesquichlorat. (same strength as nitrate of silver). Enemata of ice-water have also been used to advantage.

3. *Hæmoglobinuria.*

I. ETIOLOGY.—In this condition, the bloody color of the urine is owing to the presence of dissolved blood pigment. This condition will be produced if a large number of red blood-globules undergo dissolution within the vessels, since the kidneys very soon excrete the hæmoglobin.

In animals, this symptom may be produced by the injection into the blood-vessels of water, a weak solution of salt, the salts of the biliary acids, glycerin, by poisoning with hydrochloric or sulphuric acid, by inhalations of arsenuretted hydrogen, sulphuretted hydrogen, extensive burns of the skin, transfusion with the blood of other species, etc.

Hæmoglobinuria has been observed in man in the following conditions :

(a) Poisoning with sulphuric and hydrochloric acids, arsenuretted hydrogen, chlorate of potash, pyrogallie acid, carbolic acid, nitrobenzol, sulphate of copper ; it has also been observed in grave jaundice.

(b) Cutaneous burns, sunstroke, transfusion of lamb's blood, fatty embolism as the result of fracture.

(c) Severe infectious diseases (typhoid fever, scarlatina, diphtheria, septic fevers, intermittent fever, cyanosis neonatorum afebrilis [so-called Winkel's disease]).

This peculiar disease was observed by Winkel in an epidemic in the Dresden Maternity Hospital. The children became cyanotic and jaundiced, suffered from hæmoglobinuria, and death generally occurred after dyspnoea and general convulsions.

(d) Conditions of blood dissolution (scurvy, purpura, morbus maculosus Werlhofii, hemorrhagic small-pox).

(e) In a series of cases hæmoglobinuria is a sort of independent disease, occurring in paroxysms (paroxysmal hæmoglobinuria). The majority of cases have been observed in England and India. They always occur in males. The youngest patient was seven months old. Either no cause could be ascertained or the disease had been preceded by intermittent fever, jaundice, or rheumatism. Syphilis has been mentioned as a cause in several cases. In Neale's case the disease followed an injury in the region of the kidneys.

II. SYMPTOMS.—The urine has a bloody, not infrequently a blackish-red color ; in sufficiently thin strata it appears clear. In the fresh condition it is always acid (O. Rosenbach alone found it always alkaline). On boiling, a large coagulum of albumin forms ; it floats upon the surface, and is colored brown by blood pigment. If the coagulum is removed and boiled in alcohol, the latter absorbs the pigment and assumes a brownish-red color. Examination of the colored alcohol with the spectroscope shows the absorption bands of acid hæmatin (Fig. 66, IV.). If urine containing hæmoglobin is itself placed in front of the spectroscope, we will generally find the bands of oxyhæmoglobin and methæmoglobin, more rarely the band of methæmoglobin alone (Fig. 66, I. and II.). Still more rarely the urine contains hæmatin, but, according to Kuester and Salkowski, this occurs occasionally during the course of diphtheria.

If urine containing hæmoglobin is allowed to stand, it generally deposits a brown or brownish-red granular sediment. In many cases, red blood-globules are entirely absent ; in others they are very scanty. We generally find extremely fine reddish-yellow granules (hæmoglobin) ar-

ranged either in groups or in the form of casts. We may also find hyaline and fatty casts which are stained in places with hæmoglobin, renal epithelium containing hæmoglobin, and crystals of hæmatoidin.

The symptomatology of paroxysmal hæmoglobinuria requires somewhat more careful attention.

The excretion of the urine containing hæmoglobin occurs in paroxysms, and may continue for a few hours, more rarely several days or even weeks. As a rule, such an attack is preceded by a cold, especially if the hands or feet are affected. In some cases the attack could be produced voluntarily by taking a cold hand bath or foot bath. The attacks occur particularly in the autumn, winter, and spring, and often remain away altogether during the summer.

Three cases are reported in which the attack always occurred after a long march; in another case, after excesses in *Baccho et Venere*. In some cases, however, no cause can be ascertained.

The individual attacks generally begin with symptoms like those of intermittent fever. The patients suddenly complain of pricking in the skin, heaviness of the limbs, they yawn frequently, nausea or even vomiting is produced, with tenderness in the region of the liver and kidneys, and finally a distinct chill. This is followed by fever lasting several hours, and a gradual return to the normal temperature is accompanied by perspiration. Urticaria has been observed in several cases.

Individual symptoms are sometimes less distinct than described above, or entirely absent.

The urine now assumes the appearances of hæmoglobinuria, but in a few hours it becomes clearer, and soon returns to the normal.

Rosenbach observed albuminuria prior to the occurrence of hæmoglobinuria; more frequently the latter is followed by albuminuria of short duration. Neale detected in the urine a peculiar albuminoid which coagulated on boiling, dissolved in nitric acid, and was again precipitated on cooling.

The paroxysm sometimes lasts only a few hours. The patients then feel as if they had been beaten, and the skin and mucous membranes are unusually pale.

Kuessner first found that the serum of blood, removed by cupping the patient during an attack, had a ruby-red color—an indication that numerous red blood-globules had undergone dissolution in the general circulation. In blood drawn from the finger, Ehrlich found that the serum contained hæmoglobin, the red blood-globules were dissolved or discolored, and that they presented various shapes and were extremely small (poikilocytosis and mikrocynthæmia). Paroxysmal hæmoglobinuria must therefore be regarded as an affection of the blood-producing organs, resulting in the development of red blood-globules which possess but little resistance. Vaso-motor disturbances also probably play a part in the process.

Orsi and Murri found the kidneys enlarged and congested, the epithelium and connective tissue intact. In one of Murri's cases, there was a deposit of pigment in the renal epithelium.

A number of recoveries have been reported, especially when the disease was the result of syphilis. Under other circumstances the prognosis should be given with caution. Fresh attacks sometimes occur suddenly after the lapse of years. In rare cases several attacks have been observed in a single day.

III. TREATMENT.—The treatment should be directed against the

primary disease. In paroxysmal hæmoglobinuria the patient should be guarded against catching cold, and if the attacks recur frequently, he should be kept in bed. In syphilitic cases, anti-syphilitic measures should be adopted.

4. *Pyrocatechinuria.*

The first case was observed by Ebstein and Mueller. A child, æt. 4 months, passed a clear urine which became brown after standing, and finally assumed a Burgundy color. On the addition of liq. potassæ it became brown at once, and brownish-black on being shaken. At the same time there was an active absorption of oxygen. The urine reduced an alkaline copper solution.

Baumann showed later that pyrocatechin is found frequently, though not constantly, in normal urine.

5. *Melanuria.*

Melanuria has been observed when melanotic tumors were present in some of the organs. In rare cases the urine contains melanin and has a black color as soon as passed. As a rule, however, it is clear at first and grows dark gradually, or at once if oxydizing substances, for example, nitric acid, are added.

Primavera claims that similar symptoms develop after the internal administration of tannic acid or on its addition to the urine.

6. *Chyluria or Galacturia.*

In chyluria the urine has a milky-white color, sometimes it is yellowish-red on account of the presence of blood. In many cases a layer of cream forms upon the surface after standing. As a rule, the odor is acid, rarely slightly urin-

FIG. 68.



Filaria sanguinis hominis. After Ewald.

ous, the reaction is feebly acid or neutral, exceptionally alkaline. The urine has a great tendency to decomposition, and then smells of sulphuretted hydrogen.

In some cases the urine contains loose clots from the beginning; these have formed in the bladder and occasionally give rise to considerable disturbance in micturition. In other cases clots form only after the urine has been exposed to the air for some time.

The microscope shows very fine drops of fat, which are scattered throughout the fluid; the sediment contains red and white blood-globules. In alkaline urine we sometimes find crystals of the triple phosphates; crystals of uric acid have been noticed in a few cases. Frerichs found mature and immature spermatozoa.

On shaking the urine with ether, the fat is dissolved and the urine becomes much clearer. It always contains albumin (serumalbumin, globulin, and peptones), but no casein. Sugar is almost always absent.

In Egge's case the urine contained the following substances: urea, 2.2 per cent; uric acid, 0.03 per cent; sodium chloride, 0.35 per cent; fat, 0.687 per cent; albumin, 0.627 per cent.

2. There are two forms of chyluria, the tropical and non-tropical forms. The former (occurring in the East and West Indies, Egypt, etc.) is the result, in the majority of cases, of the presence of *filaria sanguinis hominis* in the blood. We always find the embryonal form of the animal; the mature worm has only been

discovered very recently. The former are worm-like bodies, about 0.35 mm. in length and 0.007 mm. in width (Fig. 68). The head is rounded, the tail pointed. Lewis found the parasite in the kidneys and renal vessels in this disease, but nothing is known concerning the more intimate relations between the parasites and chyluria.

In some cases of non-tropical (non-parasitic) chyluria, an abundance of fine drops of fat have been found in the blood, but nothing is known concerning the genesis of the disease.

Patients suffering from chyluria may present a healthy appearance. Pain is often felt in the region of the kidneys, and the formation of clots in the bladder may give rise to difficulty in micturition.

In many cases the disease occurs in paroxysms, continues for weeks and months, and then disappears for a long time. In the tropics, chyluria and hæmaturia have been known to alternate with one another. The urine is sometimes chylous only at certain parts of the day (generally the morning), and Mackenzie showed that the filariæ may be very abundant in the blood at night and absent during the day.

3. The diagnosis is easy; the prognosis is not very favorable. The patients should receive nourishing diet, iron and quinine; tannic acid is also recommended. In the tropics, pentaphyllum is regarded as a reliable remedy.

7. *Lipuria.*

1. In lipuria fat floats in the form of drops upon the surface of the urine. In some cases this condition is so advanced that the urine looks like fat bouillon. In Ebstein's case the drops of fat soon assumed a gray, opaque appearance, owing to the formation of innumerable needles of the fatty acids; in addition, the urine contained numerous hæmatoidin crystals.

If the drops are heated in a test-tube, acid vapors of acrolein are given off. If the urine is shaken with ether, the latter absorbs the fat.

2. In lipuria the urinary apparatus may be either intact or diseased.

It is found under the following circumstances when the urinary apparatus is intact:

a. After the administration of remedies which contain fat (cod-liver oil, emulsions, oil, and very fatty food).

b. In diabetes mellitus, in which it was attributed to the fact that the blood is unusually rich in fat.

c. In diseases of the pancreas.

d. In heart disease (Henderson).

e. In cases of poisoning (phosphorus, carbonic oxide). Perhaps this category includes lipuria occurring in acute yellow atrophy of the liver, yellow fever, jaundice, and gall-stones.

f. In fractures of bones and fatty embolism.

g. In cachexia, chronic diseases of the bones and joints, malignant tumors, protracted suppuration, pyæmia, and gangrene.

h. In pregnancy.

Lipuria may also be a symptom of diseases of the urinary apparatus (chronic parenchymatous nephritis, fatty degeneration of blood clots in pyonephrosis, in calculous cysts as the result of fatty degeneration of the pus-corpuscles and vesical epithelium after frequent pollutions, and in spermatorrhœa).

8. *Fibrinuria.*

1. In fibrinuria the urine, after standing for some time, coagulates into a gelatinous mass. It again becomes fluid after prolonged shaking. It sometimes has a reddish color, but contains very few red blood-globules.

2. Fibrinuria has been observed in epithelial cancer and in Bright's disease. It is said to occur as an endemic in Madagascar, Isle de France, and Brazil. It has also been observed after the administration of cantharides.

9. *Hydrothionuria.*

1. In this condition sulphuretted hydrogen is given off by the urine. A large amount is recognized by the odor; in other cases a piece of blotting paper, which has been dipped into a solution of acetate of lead, will turn brown or black. This

must be distinguished from subsequent decomposition of the urine and development of sulphuretted hydrogen such as occurs in urine which contains albumin or cystin.

2. In some cases the gas seems to be derived from the intestinal tract or from peritonitic abscesses which have undergone decomposition. Two cases were observed after perforation-peritonitis, another in hypertrophy of the prostate and dilatation of the rectum, another during convalescence from typhoid fever.

According to Gscheidlen, the urine contains potassium sulphocyanate which when decomposed, gives off sulphuretted hydrogen. In Ranke's case the urine always contained pus. This author believes that the development of the gas is the result of a ferment, since a few drops of the urine, when added to healthy urine, also gave rise to the development of sulphuretted hydrogen.

10. *Oxaluria.*

1. According to English writers, there is a distinct disease of nutrition which leads to increased excretion of oxalic acid in the urine. It is supposed to give

FIG. 69.



Crystals of oxalate of lime. In the middle, the ordinary octahedra, at the sides, disk, hour-glass, and dumb-bell shapes. Enlarged 275 times.

rise to serious symptoms on the part of the intestinal tract, sexual apparatus, and nervous system. German writers have always denied the existence of this disease. Its advocates confine themselves to an estimate (under the microscope) of the oxalate of lime in the urinary sediment, although it is known that this possesses no necessary relation to the amount of oxalic acid in the urine.

The crystals of oxalate of lime generally have an octahedric or envelope shape; they may also assume various other shapes (vide Fig. 69).

They dissolve in mineral acids, but not in water or acetic acid.

2. They are observed in abundance after the ingestion of certain articles of food which are rich in oxalic acid (grapes, spinach, celery, rhubarb, etc.), also after the administration of certain drugs (rhubarb, squills, valerian).

An increased production of oxalic acid has also been observed in jaundice and diabetes mellitus; this increase is inconstant in fever and respiratory disturbances.

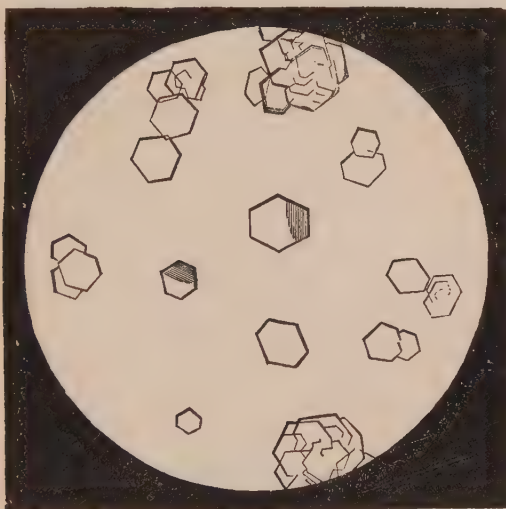
11. *Cystinuria*.

1. In cystinuria the urine contains cystin. This is precipitated in part as a sediment in the shape of six-sided tablets (Fig. 70), in part it remains dissolved in the urine until acetic acid is added. Since cystin contains a large amount of sulphur, sulphide of lead is deposited if lead oxide is added to the urine, and the latter is then boiled with liq. potassæ.

Cystinuria occurs most frequently in those individuals who suffer from cystin calculi, although the latter may be present despite the absence of cystin crystals in the urinary sediment. It may be permanent or temporary. It has been observed during the course of acute articular rheumatism, in syphilis, and in disease of the liver.

2. Cystinuria is not a frequent disease. In 1882, Ebstein succeeded in collecting sixty-one cases. Men are affected much more frequently than women (3:1). It occurs at all ages, but somewhat more frequently from the twentieth

FIG. 70.



Cystin crystals. Enlarged 275 times.

to the thirtieth years. It has been observed not infrequently in brothers and sisters.

3. The amount of urine may be normal, increased, or diminished. It is usually clear, greenish-yellow, feebly acid or neutral in reaction. When the urine is decomposed, the odor of sulphuretted hydrogen is emitted. Urea and uric acid are sometimes diminished in quantity; sulphuric acid is often increased. In Niemann's case the daily amount of cystin excreted varied from gr. 6.3-8.8. In several cases more cystin was excreted at night than during the day; in Ebstein's case the reverse held good. In the latter case, furthermore, the amount of cystin was trebled by eating lentil porridge.

In some cases the patients feel entirely well, in others they suffer from the symptoms of renal or vesical calculi.

4. The prognosis is serious because calculi are apt to form in this condition.

Nitric acid and aqua regia have been recommended in this disease. Gautain attaches the greatest importance to a chiefly meat diet.

PART II.

DISEASES OF THE RENAL PARENCHYMA.

1. *Uræmia.*

I. ETIOLOGY.—*Uræmia* is a symptom-complex which is produced whenever there are disturbances in the excretion, through the urine, of urea-producing substances in the blood. This occurs most frequently in diseases of the kidneys; also when there is a mechanical interference with the discharge of urine through the urinary passages.

As a rule, *uræmia* is preceded by greater or less diminution of diuresis. Only in rare cases is diuresis profuse or even increased; but even in such cases the *uræmia* has been preceded, for a longer or shorter interval, by a retention of the constituents of the urine.

Uræmic symptoms are observed most frequently in Bright's disease, but while the majority of patients who suffer from chronic interstitial nephritis die of *uræmia*, it is somewhat less frequent in acute nephritis, and still less frequent in chronic parenchymatous nephritis. Among the forms of acute diffuse nephritis, that following scarlatina is especially apt to be followed by *uræmia*, while this is extremely rare in nephritis following diphtheria.

It is also a frequent complication of the renal affections of cholera and pregnancy, but is extremely rare in waxy kidneys and in pure passive congestion of the kidneys.

It is sometimes associated with hydronephrosis, pyelonephritis, or nephrolithiasis, either from obstruction to the outflow of urine or because the urine undergoes ammoniacal decomposition, is absorbed, and poisons the organism. It is probable that *uræmia* sometimes follows the presence of a calculus in one ureter because this produces reflex inhibition of the functions of the other kidney.

Stasis of urine and *uræmia* sometimes follow mechanical occlusion of the ureters at their entrance into the bladder (cancer of the bladder, uterus, or rectum). Hypertrophy of the prostate and urethral stricture may act in a similar manner.

Decomposition of the urine frequently takes place within the bladder. This condition is particularly dangerous if, as in diseases of the spinal cord, the muscular coat of the bladder is paralyzed, and stagnation and absorption of the alkaline urine are thus favored.

II. SYMPTOMS.—*Uræmia* sometimes develops very suddenly and proves rapidly fatal, sometimes it is preceded by prodromata and the symptoms continue to increase in severity for many weeks (acute, sub-acute, and chronic *uræmia*).

Nervous symptoms are very frequent, and often precede other manifestations.

Many patients complain of dizziness and headache, which may be diffuse or unilateral (hemicrania). The pain is not infrequently unusually obstinate and protracted. Neuralgia sometimes develops, generally

in the trigeminus, more rarely the occipital or sciatic nerve. Anæsthesia and paræsthesia have been occasionally observed.

Spasmodic muscular symptoms are observed with extreme frequency (usually epileptiform attacks, more rarely tonic spasms of single groups of muscles). As a rule, the epileptiform seizures are preceded or rapidly followed by disturbance of consciousness.

The contractions are occasionally confined to very few muscles, and subside very rapidly. Sometimes the muscular twitchings are unilateral. In rare cases, they are followed by paralysis.

Disturbances of consciousness are extremely common, and often form the initial symptom. The patients grow apathetic, then somnolence develops, and increases to profound coma. The patients often lie for days in a somnolent or comatose condition, groaning, breathing irregularly, or presenting the Cheyne-Stokes phenomenon, and passing urine and fæces involuntarily. In other cases, unconsciousness occurs only during the convulsive attacks, so that the latter are exactly like an epileptic fit.

In some cases, these conditions are followed by delirium and even maniacal attacks.

Sight and hearing are often affected in uræmia.

Amaurosis not infrequently develops suddenly, lasts one to three days, and then disappears quite suddenly. In Bright's disease, it is sometimes found that albuminuria is absent at the development of amaurosis. This condition is not the result of retinitis albuminurica; no changes are visible in the fundus of the eye. The pupils may be dilated, and sometimes react to light; sometimes they are inexcitable.

The causes of uræmic amaurosis are unknown; some attribute it to sudden œdema of the optic nerve, others to circumscribed œdema of central parts; finally, others look upon the condition as a form of poisoning.

Auditory disturbances are partly subjective, partly objective. The patients grow hard of hearing, and complain of buzzing, ringing in the ears, etc.

Epistaxis occurs not infrequently, and is often very profuse, and checked with difficulty. This is sometimes followed by signs of blood dissolution (extravasations on the skin and mucous membranes).

Hemorrhages may occur independently in certain mucous membranes (bronchi, intestines), and sometimes constitute the direct cause of death.

Some patients have a characteristic fœtor ex ore, like that of decomposed urine. If the uræmia is protracted, the lips, gums, and tongue may be covered with sordes. Or the tongue has a grayish-white or brownish-yellow coating.

Very many uræmic patients suffer from singultus, nausea, and violent vomiting. The latter is frequent in the fasting condition and in the morning, and often consists of thin, watery masses which have a biting, distinctly ammoniacal odor.

Salivation has been observed a number of times; the saliva very often contains urea.

Diarrhœa is a frequent symptom. At times, it has a very favorable effect, at others the thin stools have a dysenteric character and pestilential odor, and are mixed with desquamated shreds of the intestinal mucous membrane.

Uræmia may also be characterized by obstinate hoarseness, the result of chronic œdema of the laryngeal mucous membrane.

Uræmic asthma occurs in some patients, often at night, and sometimes with striking regularity. This is sometimes purely nervous in its origin, sometimes the result of bronchitis.

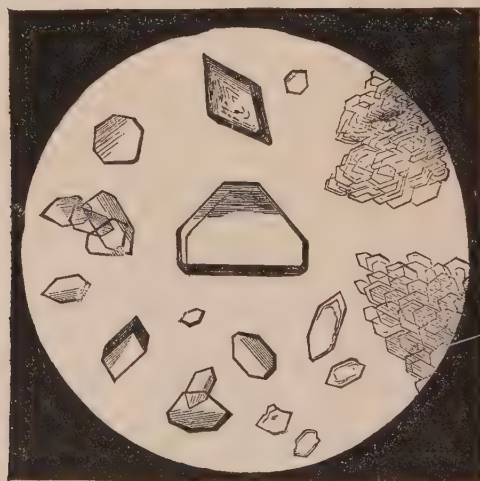
FIG. 71.



Numerous casts in the urinary sediment after an attack of uræmia, in a man æt. 27 years, suffering from primary cirrhosis of the kidneys. Enlarged 275 times.

Pulmonary oedema is an occasional symptom, and the sputum then contains not inconsiderable amounts of urea. In other cases, we find

FIG. 72.



Crystals of nitrate of urea. Enlarged 275 times.

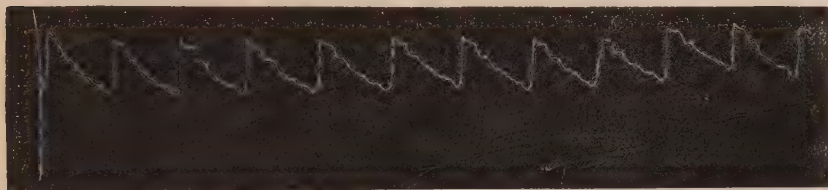
pneumonia (usually of a very flaccid character) with a fatal termination.

The uræmic process also furnishes a predisposition to inflammations

of the serous membranes; the most frequent form is pleurisy, the rarest meningitis. In one case, Bartels observed an inflammation of the knee-joint. The inflammations may be serous, purulent, or hemorrhagic in character.

As a rule, the amount of urine is diminished prior to the occurrence of uræmia; in exceptional cases it is undiminished or even increased (in the latter event, it will be found that there has been a diminution of urinary constituents). I have noticed in a number of cases of Bright's disease that, after the cessation of uræmia, an unusual number of casts appeared in the sediment of the urine, so that it would seem as if the tubules had been mechanically occluded (vide Fig. 71).

FIG. 73.

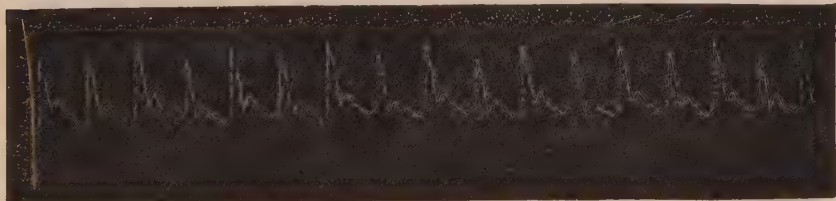


Pulse curve of right radial artery in a man æt. 57 years, suffering from contracted kidneys.

The urea retained in the body sometimes seeks an exit through other organs. The skin is occasionally covered with a white coating, especially around the hairs of the beard and axilla, which contains urea (uridrosis); on the addition of nitric acid, very characteristic crystals of nitrate of urea are formed (Fig. 72). The urea is evidently conveyed to the external integument in such cases with the aid of the perspiration.

The uræmic changes in the integument also include the often intolerable itching. More rarely we find erythematous, roseolar, wheal-like, and pruriginous eruptions; symmetrical gangrene was observed in one case.

FIG. 74.



The same, during severe uræmic coma (pulsus alternans).

The bodily temperature has a tendency to become subnormal; it may even fall below 30° .

Shortly before the onset of uræmic symptoms, the pulse is occasionally slowed; during the attack, the pulse is not infrequently accelerated, small, and often irregular. Figs. 73 and 74 give the pulse curves of a patient, Fig. 74 being taken during an attack of uræmia. It is evident that, in the uræmic pulse, the elevations of elasticity are almost entirely absent, and that the curve has become very dicrotic, in other words, that the tension of the vessel is considerably diminished. In addition, the pulse has become allorhythmic, and presents the characteristics of pulsus alternans. The diminution of vascular tension corresponds to impaired vigor of the heart's action.

In some cases the symptoms of uræmia occur unexpectedly, particularly in chronic interstitial nephritis. Under such circumstances the diagnosis of the condition may be extremely difficult.

In other cases the development of uræmia has been expected, as, for example, when diuresis has been diminished in patients suffering from renal diseases. Injudicious therapeutic measures may also cause an outbreak of uræmia. Thus, uræmia has been produced a number of times in anasarctous patients in whom the œdema was rapidly absorbed by diaphoresis, evidently because the blood was flooded with the absorbed excrementitious products.

Sometimes a single uræmic seizure alone occurs, at other times the affection lasts for weeks and months. Relapses occur very frequently.

III. NATURE OF THE DISEASE.—According to the *mechanical theory*, uræmia is the result of œdema and anæmia of the brain. According to Traube, uræmia occurs almost exclusively in those cases in which the left ventricle is hypertrophied. This favors the escape of fluid from the small arteries of the brain, and this in turn produces anæmia of the brain by compression of the capillaries and veins.

In the majority of cases this theory possesses no anatomical foundation; œdema of the brain is exceptional in uræmia.

Among the *chemical theories* of uræmia, the best known is that which attributes the condition to the retention of urea in the blood and tissues (Frerichs). Through the action of a ferment the urea is converted into ammonium carbonate, and this gives rise to the uræmic symptom-complex. According to Treitz, the urea is excreted through the gastro-intestinal mucous membrane, where it is converted into ammonium carbonate; this is then absorbed into the blood and poisons the organism.

In our opinion there is no doubt that Frerichs' theory holds good with regard to some cases. It has been proven, experimentally, that ammonium carbonate is an active poison, and produces symptoms very similar to those of uræmia; furthermore, it has been repeatedly, though not constantly, found in the blood of uræmic patients.

In very many cases, however, other excrementitious substances (potash, salts, kreatinin) seem to be the cause of uræmia.

The chemical theory is not disproved by the fact that uræmia may develop despite profuse diuresis and an undiminished amount of urea, because it has been shown that the retention of urinary constituents may remain unnoticed for a long time. Moreover, there are very great individual differences with regard to this condition. For example, anuria may last for a very variable period in different individuals before uræmic symptoms make their appearance. Thus, Willis reported a case of complete anuria (occlusion of the ureters by calculi) lasting ten days, in which uræmia did not develop.

IV. DIAGNOSIS.—We must take into consideration the etiology and make a careful examination of the urine. The condition may be mistaken for meningitis, epilepsy, cerebral hemorrhage, tetanus, trismus, delirium tremens, mania, narcotic poisoning, diabetic coma, etc.

V. PROGNOSIS.—The prognosis is grave. Apart from the fact that uræmia *per se* is a dangerous condition, we are often unable to relieve the primary affection. Death sometimes occurs suddenly with symptoms of paralysis of the heart.

VI. TREATMENT.—Diuresis should be carefully watched in patients suffering from renal diseases; as soon as it diminishes we should endeavor to increase it by the administration of diuretics.

After uræmic symptoms have developed, treatment should be strictly individualized. If the patient emits an urinous odor, the carbonate of ammonia produced by decomposition of urea should be neutralized as rapidly as possible. We may order lemonade every fifteen minutes, and, in addition, the following prescription: \mathcal{R} Acid. benzoic, gr. viiss;

camphor, gr. ss.; sacch. alb., gr. viiss. M. f. p., d. t. d. No. x. S. One powder every hour or two. I have also obtained good effects from acid. salicylic., gr. viiss. every hour.

If the pulse is small and irregular, or the sphygmograph shows deficient tension, we should order digitalis (℞ Inf. digital., gr. xv. : $\frac{3}{4}$ v., liq. kaliacetic., $\frac{3}{4}$ i.; syr. simp., 3 v. M. D. S. One tablespoonful every two hours). If heart failure develops quite suddenly, we should order vigorous stimulants, such as camphor, gr. xv.; ol. amygdal, 3 iij. M. D. S. One syringe-ful subcutaneously t. i. d.; æther sulphuric., five drops on sugar every hour, etc.

When these indications are not present, we may feel tempted to administer diuretics, but, as a rule, not much can be effected in this way. We must then endeavor to eliminate the urea in other ways, especially through the intestines and integument. Our personal preference is for drastic cathartics. ℞ Inf. sennæ comp., $\frac{3}{4}$ vi.; natrii sulphur., 3 v. M. D. S. One tablespoonful three to four times a day. ℞ Inf. colocynth., gr. xxij. : $\frac{3}{4}$ vi.; syr. sennæ, 3 v. M. D. S. One tablespoonful every three to four hours. ℞ Olei ricini, $\frac{3}{4}$ i.; gummi arab., 3 iij.; ft. c. aq. dest. q. s. emulsio $\frac{3}{4}$ v.; syr. sennæ, 3 v. M. D. S. One tablespoonful every two to three hours, etc. Four to six profuse watery evacuations should be passed every day.

Diaphoretics may be employed in addition to drastics, especially if there is marked anasarca. Pylocarpin. hydrochloric. is the only reliable diaphoretic which is employed internally; it is given subcutaneously (gr. iss : $\frac{3}{4}$ iij., one syringe-ful). But we do not possess much faith in this drug, since it materially enfeebles the heart muscle and not very infrequently causes collapse. We prefer the use of the sweat-box (Vol. I., Fig. 96). The patient may also be placed on a chair, beneath which is placed a spirit-lamp, and then covered with a woollen blanket. The following plan may also be adopted: The patient is placed in a bath at 37° R., the temperature gradually increased to 40° R. by adding warm water; he is allowed to remain in the bath for an hour, and then kept for two hours in woollen blankets.

Prominent symptoms sometimes require special treatment. Epileptiform convulsions are best controlled by profound chloroform narcosis. Venesection, leeches to the temples, ice-bag to the head, and narcotics, for example, chloral hydrat., gr. xlv. to a wineglassful of water, have also been recommended. In uræmic asthma we may make a subcutaneous injection of morphine; amyl nitrite and nitroglycerin have utterly failed in several of our cases.

2. *Ischæmia of the Kidneys.*

I. ETIOLOGY.—The integrity of the renal functions and anatomical structure is impaired as soon as the arterial supply of blood is impeded. The epithelium of the convoluted tubes presents the first anatomical changes (cloudy swelling, later fatty degeneration). The source of albuminuria, if present, must be sought in the epithelium of the Malpighian bodies; but these show slight anatomical changes, although they become unable to retain albumin in the blood if there is a deficient supply of arterial blood.

Ischæmia of the kidneys occurs in its purest state in Asiatic cholera, in which the profuse intestinal transudations cause excessive lowering of

arterial pressure. Ordinary intestinal catarrh may produce a similar effect.

Sudden, profuse losses of blood sometimes give rise to the changes in question. This is also true of those conditions in which the red blood-globules are diminished in number or impoverished in hæmoglobin (chlorosis, leukaemia, progressive pernicious anæmia, marantic and cachectic conditions).

In some cases there are changes in the renal arteries. For example, they are spasmodically contracted in lead colic, epilepsy, tetanus, suffocation, and, according to Cohnheim, in puerperal eclampsia. Fischl believes that this is also true of many painful abdominal affections, such as intestinal, renal, and biliary colic.

II. ANATOMICAL CHANGES.—The cortex is pale, light grayish-red, or, if the anæmia has lasted for some time, and fatty degeneration of the cells of the convoluted tubes has occurred, it has a grayish-yellow or even butter-yellow color. In some cases there is merely a speckled yellowish coloration.

The medullary substance may be in a condition of venous congestion, particularly in the cholera kidney. Indeed, the anatomical and clinical symptoms of renal ischæmia are best developed in cholera, so that Bartels claims that the changes are the same as those described by Cohnheim in his investigations on ligation of the renal arteries. As in the experiment on animals, it is found that the walls of the capillaries and veins, in the cholera kidney, are changed by the disturbances in circulation. They permit the escape of an abnormal amount of fluid (œdema), sometimes of red and white blood-globules, the former accumulating into infarctions, the latter passing into the tubules and urine.

In other forms of renal ischæmia the changes are generally less marked, and usually terminate in fatty degeneration. In some cases the kidneys would be regarded as anatomically intact did not the etiology and symptomatology indicate that ischæmic changes must have been present.

The fatty degeneration may also extend to the muscular fibres of the heart, the cells of the gastro-intestinal mucous membrane, hepatic cells, etc.

III. SYMPTOMS AND DIAGNOSIS.—The symptoms may be confined to changes in the urine, and consist of albuminuria and the presence of casts. If the arterial pressure is unusually low, the amount of urine diminishes, and, in cholera, complete anuria may be produced.

The albuminuria is usually slight, and the amount of albumin rarely exceeds 0.2 per cent. The casts are generally hyaline, more rarely granular or waxy. They are sometimes covered with fatty granules, round cells, or epithelium cells. In cholera the urine may also contain so-called cylindroids (very long casts of a spiral or corkscrew shape). In rare cases the casts are the sole evidence of renal ischæmia, albuminuria being entirely absent.

The urinary sediment may also contain granular or fatty epithelium cells of the urinary tubules, pelvis and ureters, round cells and red blood-globules.

The symptoms of renal ischæmia are often very fleeting. But our experience in cholera shows that the obstruction to the supply of arterial blood may not continue too long, else the condition will not be susceptible of repair.

The diagnosis is based on a consideration of the etiology. A slight

amount of albumin will distinguish the disease from nephritis. In addition, œdema of the skin is absent.

IV. PROGNOSIS AND TREATMENT.—The prognosis is unfavorable only when the kidneys have been deprived too long of a supply of arterial blood. This is rarely observed, except in cholera. The treatment depends on the primary disease.

3. *Passive Congestion of the Kidneys.*

I. ETIOLOGY.—This condition always develops when there is an obstruction to the outflow of blood from the renal veins. This is almost always the result of diseases of the circulatory or respiratory organs; in such cases the arterial supply to the aorta and its branches is also unusually slight.

Passive congestion of the kidneys is seen in uncompensated valvular lesions (most frequently in mitral stenosis), also in diseases of the heart muscle and pericardium. It is also observed in emphysema, chronic bronchitis, chronic interstitial pneumonia, more rarely phthisis, chronic pleurisy, etc.

The disease is produced much more rarely by purely local causes. Thrombosis of the renal veins scarcely possesses a clinical interest in this connection. Compression and thrombosis of the inferior vena cava, above the entrance of the renal veins, are occasionally observed.

The mechanical conditions vary according as the circulatory obstruction is cardiac or purely local in character, since, in the former, the venous congestion is associated with arterial anæmia, in the latter the arterial supply is unaffected. In the former event the urine is diminished in quantity, in the latter it remains unchanged.

We will now discuss only that form which develops in diseases of the respiratory and circulatory apparatus.

II. ANATOMICAL CHANGES.—The kidneys are unusually large, the increase being occasionally more than a third the normal size. The color is unusually dark, blackish-red or bluish-red. The capsule is generally poor in fat, tense, smooth and easily removed. Numerous distended stars of Verheyen are seen upon the surface. Extravasations are sometimes found on the surface as well as on section of the kidney. The parenchyma is very tense and resistant. A small amount of œdematous fluid can sometimes be squeezed from the section of the kidney.

If the condition has lasted a long time, atrophic changes may develop. The kidneys grow pale, their consistence increases. The cortex becomes very small in places, and we may find cicatricial retractions over which the capsule is firmly adherent.

In recent cases the changes are confined to the blood-vessels, which are unusually distended. In advanced cases there may be slight opacity and fatty degeneration of the epithelium of the tubes, particularly the convoluted tubes. Some of the glomeruli and tubes may be filled with extravasated blood, or a few epithelium cells contain pigment granules as the remains of previous hemorrhages. Hyaline casts are visible here and there in the tubes. When the condition is still more marked, the interstitial tissue is broader and particularly fibrillated, and there may even be hyperplasia of the cellular elements. The Malpighian capsules are thickened. If the fatty degeneration of the epithelium has become excessive, some of the tubes may undergo atrophy (corresponding to the atrophy of the cortex and the cicatricial retractions upon the surface of the kidneys).

III. SYMPTOMS.—Passive congestion of the kidneys is recognized

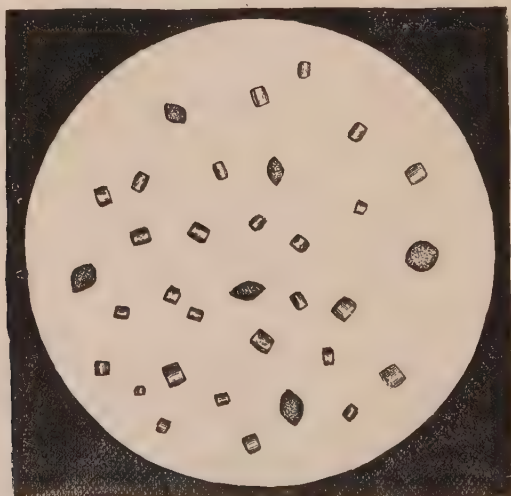
FIG. 75.



Sediment of acid urate of soda, mixed with a few oblong crystals of uric acid from a case of passive congestion of the kidneys in a woman *æt.* 45 years, suffering from mitral insufficiency. Enlarged 275 times.

only by certain changes in the urine. At the most, the patients complain of a feeling of tension and pressure in the region of the kidneys.

FIG. 76.



The same, treated with hydrochloric acid; the uric acid has crystallized into tablets, oblong and whetstone shapes. Enlarged 275 times.

The urine is diminished in quantity (to 500–300 ccm., or even less in

twenty-four hours). The color is dark, reaction very acid, and specific gravity increased to 1.030 or more. On cooling, a sediment of urates is deposited. The acid urate of soda consists of fine granules, which are dissolved on the addition of acetic or hydrochloric acid, and then, after a certain length of time, deposit crystals of pure uric acid, which have a characteristic shape (Figs. 75 and 76). The amount of uric acid is not infrequently increased.

Albuminuria may be permanently absent; in other cases a small amount of albumin is present.

In addition to the urates, the sediment contains a few round cells and epithelium cells of the bladder and urinary passages, and a few red blood-globules. The latter are rarely present in larger numbers. The urine often contains casts, which are generally hyaline, occasionally covered with fine drops of fat, more rarely with a few epithelium cells from the tubes (vide Fig. 77).

FIG. 77.



Sediment in passive congestion of the kidneys in a man æt. 30 years, suffering from mitral stenosis and insufficiency; it contained a few red blood-globules, hyaline casts, and epithelium cells. Enlarged 275 times.

The diminution in the amount of urine is attributed to the arterial anæmia of the kidneys. This also gives rise to the albuminuria, by interfering with the nutrition of the epithelium of the Malpighian bodies, so that they are no longer able to retain the albumin in the blood.

Since the stasis is not confined, in diseases of the heart and lungs, to the renal veins, symptoms of stasis are also produced on the part of other organs. The patients complain of dyspnœa, often of palpitation, more or less intense cyanosis, and œdema. As a rule, albuminuria does not develop until œdema has appeared in the lower limbs. There is often passive congestion of the liver and gastro-intestinal tract. The pulse is often very rapid, small, and irregular.

In many cases the symptoms come and go, according to the vigor of the heart's action.

IV. DIAGNOSIS.—The diagnosis is easy if we take into consideration the etiology and the urinary changes. When the urine contains a considerable amount of blood, we may be led to suspect embolism of the renal artery, if the patient suffers from heart disease. But renal embolism develops suddenly, is associated with severe pain, and not infrequently with a chill, fever, and vomiting.

V. PROGNOSIS.—The disease is not directly dangerous to life, but the prospects of recovery are unfavorable in many cases on account of the primary disease.

VI. TREATMENT.—Since the heart muscle is always affected in these cases, we should attempt to strengthen it. In addition to nourishing food and alcoholic stimulants, we must rely chiefly on digitalis. During its administration, the patient should be carefully watched. Beginning intoxication is shown by the slow, irregular pulse, a feeling of dizziness, ringing in the ears, nausea, epigastric pain, and diarrhœa. I have also repeatedly observed diplopia and hallucinations in my patients.

In addition to digitalis, we may mention caffein, convallaria majalis, and adonis vernalis (vide Vol. I., p. 125).

If the patient is very feeble, we may prescribe stimulants and tonics (℞ Camphor, gr. viiss.; gummi mimos., gr. lxxv.; f. c. aq. aurant., syr. althææ, āā. 3 vij.; aq. destil., q. s., emulsio ℥ vij. M. D. S. One tablespoonful every two hours. ℞ Inf. Valerian, ℥ ss. : ℥ v.; vin. gallic. rubri, ℥ i.; syr. simpl., 3 v. M. D. S. One tablespoonful every two hours, etc.). Diuretics are indicated if the amount of urine is unusually small. We may order carbonated waters, or, ℞ Inf. junip., ℥ ss. : ℥ v.; liq. kali acetic., ℥ i.; oxymel. scillitic., 3 v. M. D. S. One tablespoonful every two hours, etc. Cathartics should only be given if the patient is sufficiently vigorous. Diaphoretics must be employed with great caution in patients suffering from diseases of the heart and lungs, because they are apt to produce palpitation of the heart, dizziness, attacks of syncope, etc.

4. *Bright's Disease.*

(Acute and Chronic Diffuse Nephritis.)

From a clinical standpoint, we distinguish acute and chronic Bright's disease. In rarer cases we can follow the gradual transition of the acute into the chronic form; acute nephritis then constitutes the first stage, chronic nephritis the second stage of Bright's disease. In the majority of cases of chronic Bright's disease, however, the inflammation is chronic from the beginning.

Parenchymatous and interstitial inflammations of the kidneys do not exclude one another; these processes are always associated, though one may be more prominent than the other.

From a clinical and anatomical standpoint, Bright's disease may be divided into three varieties: (a) Diffuse acute nephritis; (b) Diffuse chronic parenchymatous nephritis; (c) Chronic interstitial nephritis.

These forms may develop primarily, or, in rarer cases, one develops out of another.

(a) Acute Diffuse Nephritis.

I. ETIOLOGY.—It occurs after certain epidemic infectious diseases, especially scarlatina, and is therefore frequently observed during childhood.

Climatic conditions are not unimportant. It is especially frequent at the seashore, and in cold and changeable climates.

Abuse of alcohol creates a sort of predisposition to the disease by making the kidneys less resistant. Diffuse acute nephritis is sometimes the result of a cold, and, in rare cases, of injury.

Toxic nephritis is sometimes produced by remedies which are used as drugs; for example, cantharides, squills, turpentine, copaiba, cubeb, and even potassium nitrate, carbolic acid, salicylic acid, chlorate of potash.

In some cases the injurious substances are absorbed through the skin (cantharides plaster, sinapisms, inunctions with petroleum, pyrogallac acid, mercurial ointment, carbolic acid, iodoform).

Individual idiosyncrasy plays a great part in cases of this character.

Acute nephritis often develops in poisoning with mineral acids, phosphorus, and arsenic.

Infectious diseases are a not infrequent cause of acute nephritis. It is observed most frequently in scarlatina, generally the sixteenth to twentieth day from the beginning of the disease. In some epidemics nephritis does not occur, in others it is unusually frequent. Acute nephritis is also observed after diphtheria, more rarely after typhoid, typhus and relapsing fevers, yellow fever, dysentery, malaria, meningitis, erysipelas, pneumonia, etc. It is also observed after chronic infectious diseases (syphilis, phthisis, malaria).

The connection between infectious diseases and nephritis has not been positively determined. Kannenberg found schizomycetes in the urine in infectious diseases, and assumes that bacteria make their exit through the vessels of the kidneys and thus set up nephritis. Fuerbringer, however, was unable to discover bacteria in the inflamed kidneys, so that bacteria are evidently not the sole cause of acute nephritis after infectious diseases.

The entrance of bacteria into the kidneys may be directly followed in acute septic endocarditis, in which emboli of micrococci are sometimes found in the Malpighian bodies and the intertubular blood-vessels; secondary inflammation of the renal tissue is found in the immediate vicinity of the micrococci. Acute nephritis may also follow other septic conditions: puerperal fever, abscesses, suppuration of the joints, tuberculosis of the bones, empyema, etc. We also include in this category the form of acute nephritis which occurs after extensive burns; perhaps a similar relation exists between acute nephritis and chronic diseases of the skin.

Acute nephritis is sometimes associated with blood disease (scurvy, morbus maculosus Werlhofii) and with heart diseases.

It may be secondary to inflammation of the pelvis of the kidneys, the ureters and bladder (which sometimes follow urethral stricture or acute gonorrhœa), and to inflammation of the perinephritic and paranephritic connective tissue.

In quite a number of cases no cause can be ascertained.

II. ANATOMICAL CHANGES.—The kidney sometimes appears intact to the naked eye, although the microscope reveals notable changes. If macroscopical changes are present, they are generally included under one of two forms, which may be termed the congested and the pale kidney.

The acute inflammatory, congested kidney is unusually large, and sometimes attains twice the normal dimensions. The capsule is smooth

and transparent, and is readily stripped off. The surface is very congested and the stars of Verheyen extremely distinct. The congestion is still more evident on section of the kidney. The cortex is bluish-red or blackish-red, the Malpighian bodies appear like small dots of blood. Even more marked congestion is found in that portion of the medullary substance corresponding to the territory of the arteriæ rectæ. In almost all cases we find hemorrhages, which rarely exceed the size of a pin's head, in the cortex of the kidney. The organ is unusually soft and brittle.

In the acute inflammatory, pale kidney the organ is also enlarged, but has a pale, yellowish color and the section looks opaque, particularly in the cortex. Here and there are yellowish patches which correspond to areas of fatty degeneration. Hemorrhages are usually found upon the surface and section of the kidneys, although they are sometimes entirely absent. We might infer that the pale kidney is a later stage of the congested kidney, but I have found the former in individuals who died within a few days after the beginning of the disease. I will not deny, however, that this transition may take place in some cases.

In certain cases of acute nephritis, the changes are confined to the Malpighian bodies (glomerulo-nephritis). This is noticed with relative frequency, though not constantly, in acute scarlatinous nephritis. In not a few cases accumulations of round cells are found in the interstitial tissue, especially in the immediate neighborhood of the veins and capsules. In some cases, finally, the epithelial cells of the tubes are said to be first affected. All the lesions referred to are often found associated with one another.

The following is a more detailed description of the changes which may occur in this disease :

In certain, very rapidly fatal cases, we find little more than unusual congestion of the kidneys. If extravasations form, they are found almost constantly in the Malpighian capsules or within the renal tubules. The amount of blood in the capsules may be so large as to compress and in part occlude the loops of vessels.

The predominant implication of the glomeruli is found mainly, though not exclusively, in scarlatina (glomerulo-nephritis or capsulitis). Within the capsule is found an accumulation of cells, which also compress the loops of vessels. In addition, Friedlaender found, in scarlatina, swelling and fatty degeneration of the walls of the capillaries. Hence, artificial injection of the Malpighian loops of vessels cannot be made successfully. Langhans also described proliferation and cloudy swelling of the endothelium of the blood-vessels.

The changed circulatory conditions very early affect the epithelium cells of the convoluted tubes. They become cloudy and swollen, and then undergo fatty degeneration. The inflammatory process may terminate with these changes. In some cases there is unusually active desquamation of the epithelium of the tubes—not alone the convoluted, but also the more peripheral tubes. In an examination of preparations hardened in absolute alcohol, it is found not infrequently that a more or less broad, granular layer of albumin has inserted itself between the epithelium and membrana propria of the tubules, and necessarily favors the epithelial desquamation. The interior of the tubes often contains a granular network of albuminoid substances. In some cases are found peculiar clear drops, whose chemical constitution is unknown ; other tubules are plugged by casts.

Some epithelium cells have a jagged appearance, as if they have been gnawed into. The protoplasm may be reduced to a small rim around the nucleus. Occasionally the kidney is infiltrated with numerous round cells or nuclear structures. The latter may accumulate within the tubules.

In the interstitial stroma we not infrequently find an accumulation of round cells, particularly around the Malpighian bodies and veins. They may sometimes form lymphomatous nodules as large as a pea.

In the process of repair we find that the increasing fatty degeneration of the epithelium and, in part, of the round cells in the interstitial stroma, is followed by absorption and expulsion in the urine; the epithelium cells are regenerated from emigrated white blood-globules (Axel Key).

Œdema is found very often in the skin and serous cavities; the latter not infrequently contain purulent exudations. Considerable dilatation and hypertrophy of the heart may develop in a short time.

III. SYMPTOMS.—The disease may begin in a latent manner. We have repeatedly treated children for diffuse acute nephritis, in which attention was first attracted by œdema of the skin. The patients were reported to have been entirely well, but careful questioning showed that that they had suffered from pain in swallowing (diphtheria), or diffuse redness of the skin (scarlatina). As a general thing, nephritis following infectious diseases at first runs a latent course. This is also true of rheumatic nephritis.

In other cases the disease begins with one or more chills. These may be followed by a fever for a longer or shorter period, but an apyrexial course of the disease is not infrequent. Some patients suffer from nausea and vomiting, coated tongue, anorexia, irregularity of the stools, dulness in the head. There is sometimes pain or tenderness in the region of the kidneys. The complexion soon assumes a peculiar waxy, pale, and sallow color.

More frequent than the symptoms mentioned is the development of œdema of the skin. It often appears first in the eyelids, so that they (particularly the lower lid) are distended into the shape of a sac, transparent, often slightly reddened. At first the œdema sometimes disappears at night, finally it becomes stationary. It gradually extends over the entire body, and is especially marked in the lower limbs and external genitals. It is often most marked in the most dependent portions of the body. The skin becomes pale and shining, and the pressure of the finger leaves a depression which does not disappear for some time. It is very dry, and presents little tendency to perspire. Unusual deformities of the penis are sometimes observed as the result of œdema, and may offer considerable obstruction to micturition. Marked œdema of the skin may also cause great inconvenience by rendering the patient unable to bend the swollen limbs.

When œdema is present, the urine almost always contains albumin. In those rare cases in which albuminuria is absent, Bartels assumes that the urine is excreted only by the still healthy portions of the kidneys.

Cohnheim and Lichtheim have rendered it probable that the diminution of albumin in the blood renders the cutaneous vessels abnormally permeable.

The urine is almost always diminished in quantity, and temporary complete anuria is observed not infrequently. If the patient recovers, unusually large amounts of urine are sometimes excreted, so that 3,000–6,000 ccm. may be excreted for days. Hæmaturia is present in the majority of cases, but its recognition often requires the aid of the microscope. If the individual portions of urine passed during the day are collected separately, the amount of blood present in each will often be found to vary greatly. As the patient recovers, the nocturnal urine may be free from blood, while it is still present in the urine passed during the day. In a case of acute nephritis following intermittent fever, Leube noticed hæmaturia only during the attacks of fever. But the urine always remains free of clots, a proof that we have to deal with renal albuminuria.

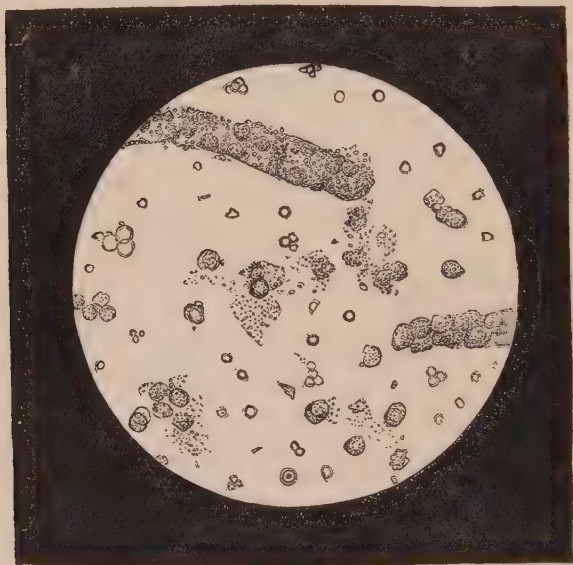
The reaction of the urine is always acid ; its specific gravity is generally increased above 1.030 (1.047 in Heller's case). As recovery begins and the amount of urine increases, the specific gravity sinks to 1.010 or even lower.

As a rule, the amount of albumin in the urine does not exceed 0.5 per cent.

If the urine contains blood and is allowed to stand for some time, it usually deposits a flocculent, brownish sediment. Its crystalline constituents are uric acid, acid urate of soda, and oxalate of lime. Its formed elements are blood-globules, casts, renal epithelium, and round cells ; in addition, we may find epithelium from the lower urinary passages.

Red blood-globules are hardly ever absent; not infrequently they are

FIG. 78.



Urinary sediment in acute (rheumatic) nephritis, in a man æt. 37 years. It contains distended and retracted, discolored red blood-globules, casts covered with round cells and epithelium from the tubules, round cells and granular detritus (fifth day of the disease). Enlarged 275 times.

so abundant as to form the chief mass of the sediment. They may be unchanged in appearance, swollen and biconvex or globular in shape; in some cases they have lost their blood pigment. They are not infrequently deposited upon casts, or aggregated into so-called blood-casts (vide Fig. 79).

Hæmatoidin crystals are found at times, though not often, in the urinary sediment. They are sometimes present during infectious diseases, although nephritis may be absent. We not infrequently find casts which have a diffuse, bloody color from imbibition of pigment.

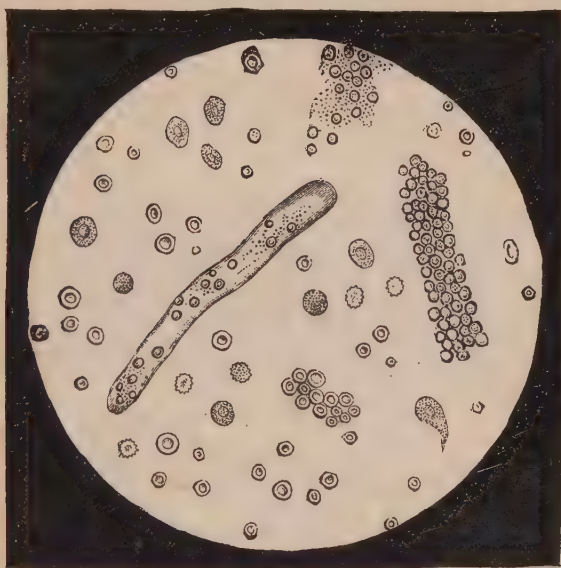
The sediment may contain all the various forms of casts—epithelial, hyaline, coarsely granular, finely granular, waxy, and blood casts.

Epithelial tubes and casts are particularly abundant in the sediment of scarlatinous nephritis. The former are composed of epithelium cells of the tubes which have been desquamated in continuity, the latter are solid cylinders covered with more or less numerous epithelium cells (vide Fig. 80). The casts are sometimes covered with round cells, drops of fat, granulo-fatty cells, urates, crystals of oxalate of lime or hæmatoidin.

In some cases numerous casts are found in each field of the microscope, in others careful search is requisite in order to find them. The presence of casts sometimes precedes the occurrence of albuminuria, in other cases they persist longer than the albuminuria.

Epithelium cells from the tubules are found either isolated or in groups, sometimes they form tubular structures, or finally they may cover a solid cast. These cells are small, usually polygonal or round,

FIG. 79.



Urinary sediment in acute nephritis-after pneumonia, in a man æt 42 years. It contains blood-casts, red blood-globules, round cells and epithelium from the tubes and lower urinary passages. Enlarged 275 times.

with a large nucleus; they are granular and, in advanced cases, more or less fatty. Their recognition is not always easy.

The round cells often present their ordinary normal shape. Occasionally we notice swelling, the formation of vacuolæ and fatty degeneration. The cells occur singly or in groups, and occasionally cover renal casts.

The changes in the urine are readily explained. The hæmaturia is partly the result of diapedesis of red blood-globules, partly of hemorrhages into the kidneys. The diminished amount of urine is explained by the serious interference with circulation, especially in the Malpighian bodies. The nutrition of the epithelium of the Malpighian bodies and convoluted tubes is impaired, and thus renders possible the passage of albumin into the urine. As the epithelium of the convoluted tubes plays a prominent part in the excretion of urea, it is not astonish-

ing that the latter is found to be diminished in amount. Sodium chloride, phosphoric acid, and kreatinin are also excreted in small quantities.

Some patients suffer from disturbances of micturition. There is a frequent desire to urinate, but only a few drops are passed at a time. Strangury may develop in cases of poisoning with cantharides, cubebs, copaiba, or even turpentine.

The circulatory apparatus often presents noteworthy changes. Occasionally we notice an unusually rapid and marked dilatation of the heart, particularly of the right side, which rapidly disappears after suitable treatment. This is observed with comparative frequency in scarlatinous nephritis. In other cases the signs of cardiac hypertrophy

FIG. 80.



Urinary sediment in scarlatinous nephritis, containing granular, hyaline and waxy casts, casts covered with epithelium, red and white blood-globules, and epithelium from the tubes. Enlarged 275 times.

also develop. The heart sometimes presents the rhythm du galop, an evidence of unusual obstruction to its action.

Riegel noticed evidences of increased tension of the radial artery. The elevations of elasticity were more distinct, those of recoil were less distinct or even disappeared (Fig. 81).

In this disease the blood contains a series of excrementitious matters which are otherwise discharged in the urine. The following changes have been observed in a number of cases: diminution of the red and white blood-globules and of the solid constituents of the blood, especially of the albumin; diminished specific gravity.

In the most favorable event the disease lasts one to two weeks. The abnormal constituents of the urine gradually diminish and disappear,

and diuresis increases. During convalescence the excretion of urine is sometimes increased, especially if extensive œdema is rapidly absorbed.

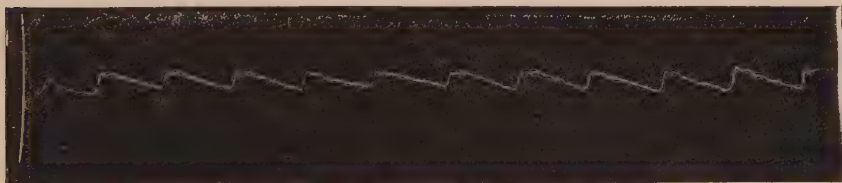
While rapid emaciation occurs during the course of the disease, recovery is often accompanied by voracious appetite and rapid and excessive development of the panniculus adiposus.

In some cases the disease lasts from four to twelve weeks, and several remissions and exacerbations may occur.

In still other cases acute nephritis passes into chronic Bright's disease and is followed by the symptoms of diffuse, chronic parenchymatous nephritis: scanty diuresis, considerable albuminuria, high specific gravity of the urine, fatty morphological elements in the urinary sediment, œdema, etc.

The course of the disease is sometimes interrupted by fatal complications. For example, serous effusion takes place into the abdominal, pleural, or pericardial cavity, pulmonary œdema develops, and the patient suffocates. Or, more rarely, sudden œdema of the glottis develops and results rapidly in death. These complications may develop unexpectedly and prove fatal within a few hours. In certain cases we find inflammatory and even purulent exudations into the serous cavities. Very rapid dilatation of the right side of the heart sometimes simulates a pericardial effusion.

FIG. 81.



Pulse curve in acute (rheumatic) nephritis, in a man æt. 37 years. Right radial artery. Fifth day of the disease.

Edema may also prove dangerous in other ways. If the cutaneous œdema is inordinate in amount, the skin sometimes ruptures in places, from which the transudation trickles incessantly; erythematous, erysipelatous, or gangrenous changes develop in the skin, and the patient dies from exhaustion, fever, or sepsis.

Uræmia constitutes the most dangerous complication. The smaller the amount of urine and of the solid constituents of the urine excreted, the greater is the danger (vide page 258).

IV. DIAGNOSIS.—The diagnosis is usually easy. In addition to the etiology, the urine presents characteristic changes; bloody urine in small amount and of high specific gravity, albuminuria, presence of blood-globules, casts, renal epithelium, and round cells in the sediment.

Acute nephritis is distinguished from passive congestion of the kidneys by the absence of causes of stasis, and by the larger amount of albumin and blood in the urine, and the greater number of casts.

Traumatic renal hemorrhage is distinguished by the previous history and by the small amount of albumin, which at first corresponds merely to the amount of blood present in the urine.

In embolic renal hemorrhage the symptoms develop suddenly with pain, chill, and vomiting, and a valvular lesion of the heart is demonstrable.

It is sometimes difficult to differentiate the disease from an acute ex

acerbation of chronic nephritis. We should endeavor to ascertain whether the signs of nephritis have been previously present.

V. PROGNOSIS.—Many patients entirely recover their health, but unexpected dangers may arise at any moment. Slight diuresis and uræmic phenomena are especially dangerous symptoms.

VI. TREATMENT.—Care should be exercised in the administration of those drugs which are known to be capable of producing nephritis. In scarlatina the patient should take lukewarm baths (28° R.) daily.

After acute nephritis has developed, the chief importance should be attached to a rational regimen. The patient must remain in bed, take a lukewarm bath (fifteen to twenty minutes) morning and evening, and drink carbonated waters. The diet should be confined chiefly to milk, with weak soup, and a little tender meat. Claret mixed with water may be allowed, tea and coffee are prohibited. Mild laxatives (inf. sennæ., pulv. liquirit. co., etc.) should be given in order to secure several passages daily. Causal treatment is indicated under certain circumstances, for example, quinine in intermittent fever, mercury in syphilis, etc.

Leeches or dry cups to the region of the kidneys have been recommended, but are not very effective.

The administration of diuretics is indicated only when the quantity of urine is unusually small, but as a general thing the inflamed organ should be kept at rest. We should restrict ourselves to acetate or bitartrate of potash, nitrate of potash, and other mild diuretics.

If the cedema becomes excessive, we may resort to vigorous diaphoresis (vide page 263).

Pilocarpine hydrochlorate (gr. iss. : 3 iij., one-half syringeful subcutaneously) has been employed a good deal lately, but this drug produces annoying salivation, and in many patients gives rise to vomiting, and even dangerous weakness of the heart. The vomiting can often be prevented by giving a little brandy half an hour before the injection, and adding to the pilocarpine gr. $\frac{3}{4}$ morphine.

Very marked cedema of the skin may require local treatment. Gold plated or nickel-plated needles of a hypodermic syringe may be inserted into the skin, fastened by means of adhesive plaster, and the free end armed with a thin rubber tube, which passes into a vessel into which the fluid constantly trickles.

After the symptoms have subsided, the patient should be kept quiet and guarded against exposure for a considerable time in order to avoid a relapse.

b. Diffuse Chronic Parenchymatous Nephritis.

I. ETIOLOGY.—As a rule, this disease gradually develops as a primary and independent disease, more rarely it follows a diffuse acute nephritis. This occurs with relative frequency when the acute stage developed as the result of cold, scarlatina, intermittent fever, syphilis, or suppuration.

Diffuse chronic parenchymatous nephritis generally develops between the ages of twenty and fifty-five years. It is rare in childhood, but Bradley reports a case in which the symptoms were presented by a child æt. four months, suffering from congenital syphilis. Men are affected more frequently than women; it is more common in districts in which the weather is inclement and changeable.

In not a small number of cases no cause can be discovered. Others have been attributed to the following causes:

- a. Cold, exposure, living in damp, mouldy apartments.
- b. Losses of vital fluids and suppuration (phthisis, chronic intestinal affections, gastric ulcer, suppuration of the bones, skin, and joints).
- c. Malaria and syphilis. The development of the renal disease depends in part on the character of the epidemic of malaria, and also upon the local conditions. A stay in a malarial region may be sufficient to produce chronic parenchymatous nephritis.
- d. The effects of abuse of alcohol and excessive use of mercury have not been proven with certainty. Albutt believes that depressing emotions act as a cause.

II. ANATOMICAL CHANGES.—The kidneys are very large (sometimes two or three times the normal dimensions) and their weight is considerably increased. The capsule is usually removed with facility. The organ has a butter-yellow or grayish-yellow color, distended stars of Verheyen are seen in only a few spots. On section of the kidney it is found that the enlargement affects chiefly the cortex. There is a striking contrast in the color of the cortex and medulla, the former having a yellowish color, the latter being red and congested. In the cortex we will find yellowish streaks and dots imbedded in a part which has more of a grayish tinge; the former correspond to fatty tubules and Malpighian bodies.

On microscopical examination the changes are hardly ever confined to the epithelium of the urinary tubules. Interstitial changes are never absent, though they may be very slight. The most important changes consist of fatty degeneration of the epithelium cells, either chiefly or exclusively in the convoluted tubes. The cells are filled with a greater or less number of drops of fat; in places they form granulo-fatty cells, in others they dissolve, and the lumen of the tubes is then filled with drops of fat. Some of the tubules contain a fine network of albuminous substances, others contain casts, in places they present varicose dilations.

The Malpighian bodies are affected in the majority of cases. The epithelium cells are not infrequently swollen, proliferated and, in places, in a condition of fatty degeneration. Some capsules are partly filled with a finely granular albuminoid substance; the loops of vessels contain very little or no blood. Nuclear proliferation, fatty degeneration, and, in a few cases, a peculiar swelling are noticeable in the walls of the vessels.

The connective-tissue stroma of the kidneys is slightly swollen in some parts, in others there is proliferation of the cellular elements. We sometimes notice accumulations of round cells, either in the immediate vicinity of the Malpighian bodies or around the tubules. The stroma occasionally contains drops of fat which are evidently the result of fatty degeneration of cellular elements.

The large white kidney not infrequently undergoes retrogressive changes, and is thus converted into the secondary retracted kidney. If fatty epithelium is removed from the tubules, the latter collapse, their membrana propria thickens, the inner surfaces adhere to one another and complete obliteration is produced. Similar processes take place in the Malpighian bodies: thickening of the capsule and retraction of its contents; calcification in some of the capsules. The interstitial proliferations assume the upper hand and extend to the surface of

the kidney, the round cells are converted into connective-tissue cells, cicatricial retractions develop upon the surface and form adhesions to the capsule, the surface becomes uneven and nodular. The individual nodules are larger than in primary retraction of the kidneys, and the formation of cysts is observed only in exceptional cases. The organ retains its butter-yellow color, in primary retraction of the kidneys it has a reddish appearance.

If the reddish-gray bands of newly-formed connective tissue become excessive, the kidney assumes a speckled appearance.

Leyden showed that there are large white kidneys with an almost perfectly smooth surface and no retraction, but which present the most marked interstitial changes.

The following changes are found in the other organs: œdema of the integument, transudations (not infrequently exudations) into the serous cavities, often œdema and inflammation of the lungs, sometimes necrotic changes in the intestines; cardiac hypertrophy (unilateral or bilateral) is by no means infrequent.

Southey furnishes the following statistics, embracing 141 autopsies:

Dropsy,	106	(75 per cent)
No dropsy,	35	(65 ")
Heart normal,	67	(47.5 ")
Valvular lesion and cardiac hypertrophy,	39	(27.6 ")
Simple cardiac hypertrophy,	27	(19 ")
Phthisis,	11	(8 ")
Death from uræmia,	38	(27 ")

III.—SYMPTOMS.—In some cases the clinical history begins with the symptoms of acute nephritis which pass very gradually into those of chronic Bright's disease. But as a rule the disease begins very slowly, and is either discovered accidentally on examination of the urine or the patients complain of weakness, anorexia, shortness of breath, and especially œdema of the skin.

œdema of the skin is one of the most frequent symptoms, and develops in the same way as in acute nephritis (vide Vol. II., page 271). It is often associated with transudation into the pleural, pericardial, or peritoneal cavities. Pulmonary œdema is a not infrequent cause of death, more rarely this follows sudden œdema of the glottis.

The patients generally present an unusual pallor of the integument.

As a rule, the quantity of urine is diminished and it may sink to 300–200 ccm. There are often great variations in the amount of urine on successive days. The urine is dark, usually of a yellowish-red color. It is generally cloudy, even after standing for some time, because the urates remain suspended in the albuminoid fluid. The specific gravity is generally above 1.020, occasionally it exceeds 1.040. The reaction is acid. The urine contains a large amount of albumin (sometimes more than 5 per cent). The daily urine may contain more than 20 gm. albumin. The sediment is usually abundant. It contains casts, which are long and narrow at the beginning of the disease, later they become broader, shorter, more brittle. The casts may be hyaline, finely or coarsely granular, or peculiarly shining and waxy. Coarsely granular and waxy casts usually do not appear until a late period of the disease and then, as rule, the urine contains all the varieties of casts (vide Fig. 82). They are covered not infrequently with fine drops of fat, sometimes with epithelium cells from the tubes. Granular cells and fatty

renal epithelium are also found free in the sediment, sometimes in very large numbers. In addition, more or less numerous round cells, and here and there a few red blood-globules.

Ackermann reported a case in which the urine contained hardly any casts during the last thirteen weeks of the disease. At the autopsy the pelvis of the kidneys contained 6-8 gm. of a dark, thin mucoid fluid, which was composed of an enormous number of casts.

FIG. 82.

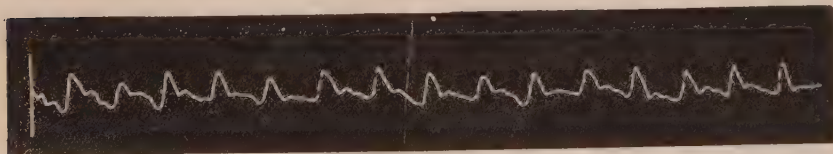


Urinary sediment in diffuse chronic parenchymatous nephritis, containing hyaline and granular casts covered with drops of fat, granulo-fatty and fatty cells. Enlarged 275 times.

The percentage of urea is sometimes increased, but the amount excreted in twenty-four hours is usually normal or less than normal. Sodium chloride, phosphoric acid and kreatinin are diminished. Brunton and Power detected, in one case, two ferments resembling ptyalin and trypsin.

Local examination of the kidneys usually furnishes negative results.

FIG. 83.



Pulse curve of right radial artery in a woman *æt.* 40 years, suffering from chronic parenchymatous nephritis.

The patients generally complain of anorexia. Eructations and vomiting are frequent. The pulse is often small and soft. Cardiac hypertrophy is much rarer than in chronic interstitial nephritis; if hypertrophy is absent, the pulse curve sometimes indicates diminution of vascular tension (vide Fig. 83). As a rule, the bodily temperature is normal. Retinal changes are rarer than in interstitial nephritis.

The disease generally lasts many months and even years. Recovery is possible, but takes place only in exceptional cases. Relapses often occur after cold, exposure, and overwork.

Acute exacerbations are occasionally noticed. The urine becomes bloody and assumes the characteristics of acute nephritis. In certain cases such attacks are unusually frequent.

As a rule, the disease terminates in death. In some cases this is the result of intercurrent inflammation of the serous membranes or lungs, in others, of excessive transudation into the serous cavities. Sudden œdema of the glottis and extensive bronchitis may cause danger of suffocation. Cutaneous œdema sometimes gives rise to fatal erysipelas and gangrene of the skin. Some patients die from marasmus; this is often hastened by the production of stinking, muco-purulent diarrhœa, which occasionally contain shreds of tissue from the intestinal mucous membrane. Death is less frequently the result of uræmia or cerebral hemorrhage.

If the disease is protracted and marked, secondary retraction of the kidneys takes place—the symptomatology undergoes a notable change. The quantity of urine increases, though not above the normal; the specific gravity diminishes (1.012–1.016), the amount of albumin and sediment diminishes, and cardiac dilatation and hypertrophy make their appearance. The latter develop at first in the left heart, and later may extend to the right ventricle.

IV. DIAGNOSIS.—The disease is usually distinguished, without much difficulty, from acute nephritis and chronic interstitial nephritis.

In acute nephritis, we find hæmaturia, an acute onset, and slighter œdema. We may remain in doubt in cases in which chronic parenchymatous nephritis undergoes an acute exacerbation.

In chronic interstitial nephritis, œdema is absent or very slight, the amount of urine is increased, the specific gravity low, albuminuria slight. In addition, we find hypertrophy and dilatation of the left heart, frequent occurrence of retinitis albuminurica, and frequent death from uræmia or cerebral hemorrhage.

It is more difficult to make a differential diagnosis between secondary retraction of the kidneys and primary, chronic interstitial nephritis. In the former, there is marked œdema in the previous stage, the amount of urine does not exceed the normal, the specific gravity is somewhat higher than in chronic interstitial nephritis, and albuminuria is more marked; the sediment also contains a greater number of fatty elements.

In many cases the differential diagnosis between chronic parenchymatous nephritis and waxy kidneys is impossible. It can only be made when the smooth enlargement of the liver and spleen indicates that the large abdominal glands have undergone extensive waxy degeneration.

V. PROGNOSIS.—The prognosis is almost always unfavorable. The fatal event will occur so much more rapidly the more extensive the œdema and the smaller the amount of urine. The danger of a sudden unfavorable termination is rendered greater, owing to the fact that the patients exhibit a predisposition to inflammation of important organs.

VI. TREATMENT.—Prophylaxis includes surgical operations to relieve suppurating processes, etc., and the treatment of intermittent fever or syphilis, from which the patient may be suffering.

The regulation of the diet is an important feature. Milk and buttermilk cures are specially recommended (vide Vol. II., page 246).

We attach great importance to baths and diaphoresis, but it should be remembered that, if the patient is suffering from extensive oedema, profuse sweating occasionally causes an outbreak of uræmia, because an excessive amount of toxic substances is suddenly absorbed by the blood.

Immermann obtained good effects from large doses of liq. kali acetic. (3 iij.—vi. daily).

In other respects the treatment is the same as in acute nephritis, except that preparations of quinine and iron are often beneficial.

c. Diffuse Chronic Interstitial Nephritis.

(Primary, Genuine Retraction of the Kidneys.)

I. ETIOLOGY.—Very few cases of this disease occur during the first decade of life. The earliest case occurred in a child æt. one and a half year (Buhl). The disease is also rare in the second and third decades, and is most common from the fortieth to the sixtieth years. One form of the disease may be termed senile retraction of the kidneys, and is associated with general arterio-sclerosis.

Men are affected more frequently than women. The disease is more common among the poorer classes, especially those who are much exposed to the vicissitudes of the weather. It is more frequent in cold and damp regions and along the coast than in inland regions which possess a more uniform, mild climate.

Among the special causes is the abuse of alcohol, but the potency of this factor was formerly overestimated.

Among the toxic forms of chronic interstitial nephritis are the lead and gouty kidneys. The former occurs in chronic lead poisoning, the latter evidently depends upon changes of nutrition, especially the retention of uric acid.

An outbreak of the disease is sometimes occasioned by infectious diseases, for example, intermittent fever and syphilis. It may also follow suppurations and losses of vital fluids of all kinds. It is occasionally secondary to chronic diseases of the lower urinary passages; for example, gonorrhœa, cystitis, and calculi.

Finally, a certain influence is exerted by heredity. In a family under my observation, the grandmother, mother, two sons, and a daughter suffered from chronic interstitial nephritis.

II. ANATOMICAL CHANGES.—There are two forms of the disease, the senile or arterio-sclerotic form, and primary retraction of the kidney in the stricter sense.

The former is the result of senile changes and takes its origin in arterio-sclerosis, especially of the renal arteries and its branches. This interferes with the blood supply and nutrition of the kidneys, causes atrophy of those glomeruli whose vessels are narrowed or occluded, and then is followed by atrophy and collapse of the tubules. The latter become filled here and there with colloid masses and are converted into cystic spaces. These changes develop usually in patches. The interstitial tissue is often very little changed. In many cases proliferation of connective tissue takes place only in places. The kidneys are small and the surface contains cicatricial depressions, but do not possess the leathery, firm consistence of primary retraction of the kidneys. The cortex is very much diminished in size, and in places is reduced to a height of only one to two mm.

In primary (genuine) retracted kidneys the organ is usually surrounded by an excessive amount of fat. The capsule is thickened, either diffusely or in patches, and is so firmly adherent in many places that it cannot be removed without tearing the kidney. The latter is unusually small, and its size and weight may be diminished by half. The surface of the kidney has a brownish-red or reddish-brown color; it is uneven and nodular. The prominences are sometimes uniform, sometimes they vary greatly in size (0.5–5.0 mm.). They consist of relatively unchanged renal parenchyma, while the retracted parts between them are composed of the hyperplastic interstitial connective tissue. The nodules have a reddish-brown, the depressions a grayish-red color.

The consistence of the kidney is hard and leathery. The cortex is unusually narrow; in places it may be only one mm. in width. The medullary substance is diminished in size to a less extent. Cystic cavities, with colloid or puruloid contents, are found sometimes upon the surface of the kidney, still more frequently about the middle of the medullary substance.

The pelvis of the kidney is unusually dilated, and sometimes presents catarrhal changes.

On microscopical examination, we find an increase of the interstitial (intertubular) connective tissue. In many places large parts of the normal tissue have been destroyed and are replaced by connective tissue. In the oldest foci this presents a fibrillated structure, in more recent ones we find a homogeneous basement substance with round cells or stellate connective-tissue cells.

In many places we can recognize the gradual destruction of tubes and Malpighian bodies. In the peripheral portions of the connective-tissue new-formation are found tubules of unusually small size. Their membrana propria is thickened and surrounded by an unusually dense accumulation of cellular elements. The tubules sometimes contain fatty epithelium or shrivelled remnants of the cells, or they are entirely devoid of epithelium. In places they contain casts or a branching network of albuminoid substances.

In some places there is a localized obliteration of the urinary tubules, which results in cystic spaces, with mucoid, more rarely puruloid contents, from colloid degeneration of the epithelium. This contains leucin, often tyrosin; in one case Rosenstein found paralbumin.

Some of the cystic cavities are the result of destruction and constriction of the Malpighian bodies. But the large portion of the Malpighian bodies is destroyed by gradual retraction. The capsules are found inclosed in laminated connective-tissue bands which are rich in nuclei and which gradually narrow the cavity. The loops of vessels also undergo fibrous transformation, occasionally a peculiar hyaline degeneration.

Opinions still differ as to whether the epithelium of the tubes or the interstitial tissue is affected primarily.

In certain forms of the disease it is probable that the process begins with changes in the vessels. Indeed, it is probable that the vascular changes in the kidneys are a part, in certain cases, of a general disease of the vessels. It must be remembered, however, that the vascular changes are sometimes secondary to interstitial changes.

The vascular changes in question may start in the tunica intima of the smaller arteries. A very lively development of epithelioid cells, with a few round cells, takes place between the fenestrated membrane and the endothelium, and pushes the latter into the lumen of the vessel, so that this is more or less completely occluded (endarteritis obliterans).

Other vessels present the changes described by Gull and Sutton as arterio-capillary fibrosis. Peculiar hyaline masses, which are slightly striated in places, are first deposited in the tunica adventitia or the muscular coat. These masses have a waxy appearance, but do not react to the tests for amyloid. This hyaline change is associated with more or less considerable stenosis of the vessel. Johnson has also observed a thickening of the muscular coat.

All three forms of vascular lesion may be present at the same time. In places we also find periarteritic changes.

Thoma found, on injecting the vessels, that they were unusually permeable.

The anatomical changes are sometimes more marked in one kidney than in the other, and even in the same kidney the lesions may be more advanced in one place than in another. In the less affected parts, the Malpighian bodies are often unusually large (compensatory hypertrophy).

The changes in the so-called red, contracted kidney, just described, are almost identical with those of the gouty kidney. In almost all cases the latter contains uric-acid infarctions which form grayish-white streaks in the pyramids, and similar dots and patches in the cortex. They consist of urates, and are situated at first within the tubes, later also in the intertubular connective tissue.

Some forms of lead kidney are exactly like the gouty kidney, when the lead poisoning has given rise to lead gout and this to renal disease.

In some cases interstitial changes in the kidneys are associated with waxy degeneration.

Morbid processes are generally found in other organs, partly of a primary, partly of a secondary nature.

Primary changes occur, in the red contracted kidney, in the circulatory apparatus. The vessels of the body present the changes described above in the renal arteries; Leyden noticed them even in the coronary arteries of the heart.

The heart muscle, as a rule, is in a condition of hypertrophy and dilatation, more rarely the former alone; sometimes the left ventricle alone, sometimes both sides of the heart are affected. Leyden also found myocarditic foci, associated with obliterated vessels. The heart is not infrequently fatty in places.

Changes in the endocardium—thickening, calcification, occasionally recent inflammation—are very common.

Transudations, exudations, and thickenings may be found in the pericardium.

Arterio-sclerotic changes are not unusual in the large vessels.

The pleural cavity may contain transudations or exudations. The lungs often show œdema or flaccid inflammation. Bronchitis may be present, occasionally œdema of the glottis.

The peritoneum not infrequently shows inflammatory changes; transudations may also be present. The gastro-intestinal tract often presents the signs of catarrh, occasionally of necrotic and ulcerative changes. Cirrhotic changes in the liver have often been described.

Bartels emphasizes the frequent occurrence of thickening of the bones of the skull. Opacity and thickening are often found on the dura mater and arachnoid, rarely evidences of meningitis.

Anæmia and œdema of the brain are occasionally noticed. Old or recent cerebral hemorrhages are found not infrequently.

Southey furnishes the following statistics of 358 cases:

No dropsy at death,	272	(76 per cent)
Dropsy,	86	(24 “)
Cardiac hypertrophy,	241	(67 “)
Valvular lesions and hypertrophy,	96	(27 “)
Cerebral hemorrhage,	79	(22 “)
Phthisis,	40	(11 “)

III. SYMPTOMS.—The manifest symptoms of chronic interstitial

nephritis refer chiefly to the condition of the urine, the circulatory apparatus, and ocular changes.

But not infrequently the autopsy discloses chronic interstitial nephritis, although hardly any symptoms have been present during life.

Many patients complain of nothing beyond annoying palpitation of the heart, which sometimes occurs spontaneously, sometimes after mental or bodily exertion. This symptom is suspicious if the pulse is hard, if the patients are young, and have not suffered from articular rheumatism. The suspicion grows almost to a certainty if examination of the heart shows a lifting-apex beat, displaced to the outside and below, and the heart sounds are clear, but the second (aortic) diastolic sound is intensified.

In other patients the first symptom is headache, particularly hemicrania. This is uræmic in character and often accompanied by vomiting, which is particularly apt to occur early in the morning.

Some patients suffer from the symptoms of gastro-enteritis. According to Hlava and Thomayer, gastric symptoms occur only in those patients in whom the autopsy discloses interstitial gastritis.

Not infrequently the patient first comes under the care of the oculist on account of increasing weakness of sight. Examination shows retinal changes which alone are almost characteristic.

The disease sometimes gives rise to obstinate relapsing hoarseness, the result of chronic œdema of the laryngeal mucous membrane.

Protracted auditory disturbances (impaired hearing, tinnitus aurium) should also awaken a suspicion of chronic interstitial nephritis; this is also true of profuse, relapsing epistaxis.

Some patients complain of great thirst and increased excretion of urine.

The first symptoms sometimes appear in the form of epileptiform spasms or cerebral hemorrhage.

Chronic interstitial nephritis is so often overlooked because œdema of the skin is either entirely absent or very slight. This symptom is absent, as a rule, on account of the increased diuresis. It makes its appearance when diuresis becomes scanty, either on account of the advanced changes in the kidneys, or because the vigor of the heart's action is impaired as the result of fatty degeneration of the heart muscle, more rarely of pericarditis. We have also noticed the development of œdema if the patients have exposed themselves to inclement weather, rain, or draughts.

The urine is excreted in abnormally large quantities (six times the normal amount in Bartels' case), but presents considerable variations on different days. The amount passed at night is often greater than that passed during the day. Constant diminution of diuresis is not a favorable sign.

The color of the urine is generally light-yellow; it is often very frothy. The reaction is almost always acid; an alkaline reaction is almost always the result of medication. The specific gravity is diminished. In one case I found it 1.002, it is rarely above 1.010 to 1.012. The specific gravity of the daily urine is usually higher than that of the nocturnal urine. It is a noticeable fact that the specific gravity increases very little during intercurrent febrile conditions.

The urine contains albumin in small amounts. The amount of albumin passed in twenty-four hours is often one to four grams, but does not often exceed this figure.

Albumin is occasionally absent in certain portions of the urine, especially in that passed at night; it is sometimes found in the daytime only after bodily exertion or mental excitement. The albuminuria sometimes disappears entirely for weeks, and even months. In two cases I noticed that albumin was present only for a short time before an uræmic convulsion, and persisted for a few days after the attack.

In rare cases, albuminuria remains constantly absent. In some instances the characteristic ophthalmoscopic appearances alone rendered a diagnosis possible, in others the changes in the circulatory apparatus are also present.

Urinary sediment is sometimes almost entirely absent. In other cases there is a scanty grayish-white dust-like sediment, which usually contains only a few casts. As a rule, they are very long and narrow, hyaline, and occasionally covered with a few drops of fat (vide Fig. 84.) A

FIG. 84.



Urinary sediment in chronic interstitial nephritis, in a man æt. 27 years; containing a few hyaline casts. Enlarged 275 times.

few casts may be covered with epithelium cells from the tubules or crystals of oxalate of lime. Broad, granular casts are rarely, waxy casts are never found.

In one of my patients the casts were unusually long and remarkably abundant. The sediment formed a layer more than one cm. in height, and contained hardly anything but hyaline casts; they were visible to the naked eye as thin threads, which were almost 0.5 cm. in length. The sediment appeared after an uræmic convulsion, which was probably the result of occlusion of the urinary tubules by the casts.

The sediment is very poor in cellular elements—a few epithelium cells from the lower urinary passages, a few round cells, and occasionally a red blood-globule. The latter increase in number during acute exacerbations.

The percentage of urea is always diminished, but the daily amount is often normal on account of the large amount of urine passed; indeed, it is sometimes increased (sixty grams in Tellegen's case). The amount of urea in the urine sometimes diminishes very considerably and accumulates in the blood; occasionally it is deposited on the integument as a whitish-gray coating (uridrosis).

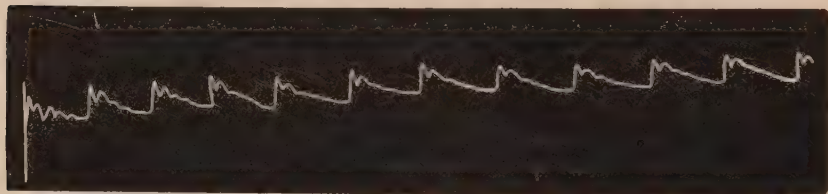
The amount of uric acid is almost always diminished; Hoffman noticed diminution of kreatinin. The excretion of sodium chloride is unchanged, the phosphates and sulphates are usually diminished.

The left ventricle is almost constantly hypertrophied and, as a rule, dilated; the right ventricle is often similarly affected.

Hypertrophy of the left ventricle is recognized by the lifting apex beat and the intensification of the second (diastolic) aortic sound. In dilatation of the left ventricle, the apex beat is broadened and displaced to the outside and inferiorly. Hypertrophy of the right ventricle is shown by the unusually vigorous movement of the lower part of the sternum and intensification of the second pulmonary sound. Dilatation of this cavity is shown by the extension of the greater (relative) cardiac dulness to the outside of the right edge of the sternum or of cardiac resistance more than two cm. beyond the right edge of the sternum.

The radial pulse is often unusually tense and hard, and this in itself may arouse a suspicion of the disease. Basch found the pressure in the

FIG. 85.



Pulse curve of right radial artery in chronic interstitial nephritis, in a man æt. 27 years.

radial artery to be 240 mm. of mercury (normally 160 mm.). In the pulse tracing, the elevations of elasticity are unusually distinct; those of recoil are less marked (vide Fig. 85).

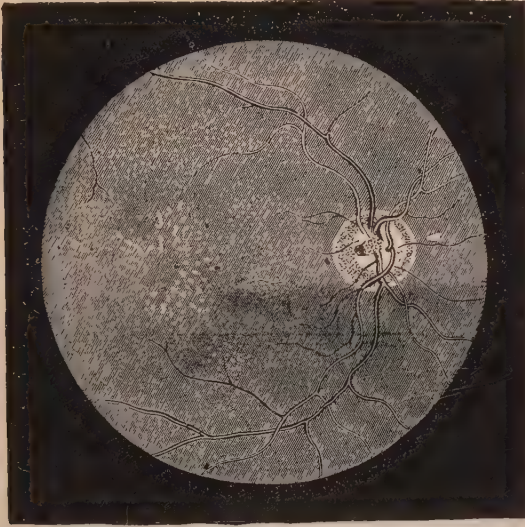
Hypertrophy of the heart is often associated with palpitation, rush of blood to the head, frequent epistaxis, and predisposition to cerebral hemorrhage. Traube also attributed the retinal changes to cardiac hypertrophy.

Traube believed that the destruction of numerous vessels in the kidneys caused increase of the blood pressure, and secondary hypertrophy of the heart. But it must be remembered that much larger vascular tracts may be destroyed, for example, in amputations, without giving rise to cardiac hypertrophy. Furthermore, this lesion may also develop in acute and chronic parenchymatous nephritis, in which narrowing of the vessels is not a marked feature. Bright attributed the hypertrophy to the retention of excrementitious matters in the blood. These changes may be very complicated. For example, changes in the peripheral arteries which cause narrowing of the lumen of the vessels will also exert an effect upon the heart. Indeed, we are perhaps justified in assuming that, corresponding to the hyperplastic processes observed by Johnson in the muscular coat of the small arteries, similar hyperplastic changes may take place in the heart muscle.

In rare cases, acute endocarditis develops during the course of the disease. The rhythm du galop, which Potain regards as important in the diagnosis of chronic interstitial nephritis, is also observed in numerous other affections.

The ocular changes cannot be diagnosed from the subjective symp-

FIG. 86.



Fundus of the eye in retinitis albuminurica. White patches in stellate arrangement around the macula lutea. After Jaeger.

toms. The latter include impairment of vision, flashes of light before

FIG. 87.



Retinitis albuminurica with irregularly scattered white patches. After Magnus.

the eyes and metamorphopsia, *i. e.*, objects appear interrupted or dis-

torted. In some cases there is temporary amaurosis, which is usually dependent on uræmia, and not upon anatomically demonstrable changes.

Among the characteristic retinal changes are fatty degeneration and sclerosis. These changes also occur, though much less frequently, in acute and chronic parenchymatous nephritis, even in waxy kidneys; also in diabetes mellitus and other diseases. The retinal changes are observed in six to seven per cent of all cases of chronic interstitial nephritis.

The ophthalmoscopic appearances in retinitis albuminurica are easily recognized (vide Figs. 86 and 87). The retina contains whitish patches which sometimes surround the optic papilla more or less completely, sometimes are scattered through the retina, particularly around the macula lutea.

The patches do not all present the same anatomical structure. The

FIG. 88.



Choked disk in contracted kidneys. After Magnus.

larger ones, especially those around the optic disk, are the result of the formation of fat granules, especially in the two granular layers and the intergranular layer of the retina, while the smaller ones (around the macula) are the result of sclerotic hypertrophy of the nerve-fibres. The process may undergo complete recovery.

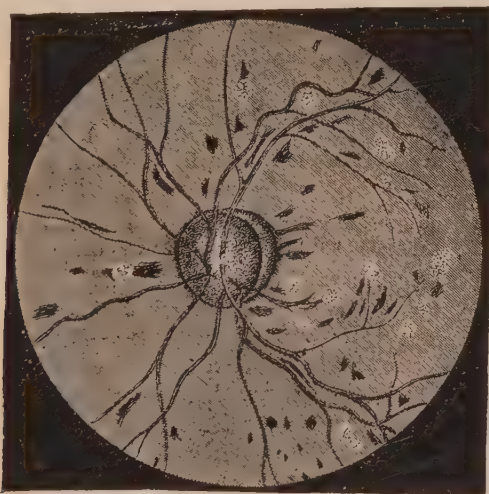
In some cases we find choked disk (vide Fig. 88) which cannot be distinguished from that occurring in cerebral tumors. The papilla is prominent and projects forward, the retinal veins are unusually wide and sinuous and ascend quite vertically from the middle of the papilla, the arteries are very narrow, the disk is reddened, its periphery grayish. Other changes in the retina may be absent. This lesion may also recover, but in some cases it seems to be followed by atrophy of the optic nerve.

Retinitis apoplectica may also develop in chronic interstitial neph-

ritis, more rarely in other forms of nephritis (vide Fig. 89). The retina has an opaque, grayish-red color, the vessels are obscured in places, and more or less numerous old and recent hemorrhages are visible, usually near the larger vessels. They may be absorbed and leave whitish patches, which should not be mistaken for fatty and sclerosed portions of the retina.

In the majority of cases there is a combination of the three conditions just described. In addition, the following lesions have been observed: *a.* embolism of the retinal arteries; *b.* separation of the retina; *c.* discoloration and changes in the pigment epithelium of the retina; *d.* choroiditis; *e.* hemorrhage into the vitreous humor, opacity, increase of cells, formation of threads of fibrin in the vitreous humor; *f.* hemorrhages under the conjunctiva and into Tenon's sheath, with secondary exophthalmus.

FIG. 89.



Retinitis apoplectica albuminurica. After Magnus.

Traube attributed the retinal changes in great part to the cardiac hypertrophy, but the former are sometimes found in the absence of the latter. It is probable that the impoverishment of the blood as regards albumin changes the constitution of the blood to such a degree as to produce changes in the retinal vessels and secondary disturbances of nutrition in the retinal tissue.

The patients are usually very pale. The integument is thin, dry, and presents very little tendency to perspire. Obstinate eczema and occasionally intolerable pruritus are sometimes observed.

The muscles gradually diminish in size and firmness, but emaciation rarely develops with such rapidity as in parenchymatous nephritis. Sexual desire often disappears very early; spermatozoa are found occasionally in the urine.

The appetite is usually impaired, thirst is increased. The patients complain not infrequently of eructations and vomiting. In uræmia the vomiting becomes very violent and obstinate, and sometimes lasts many weeks. The bowels are usually constipated, but in uræmia we some-

times notice violent diarrhœa of a dysenteriform character. Bartels saw a fatal termination in one case from intestinal hemorrhage.

The respiratory apparatus exhibits a tendency to inflammatory changes. The patients are often hoarse and suffer a good deal from bronchitis.

The disease may last many years (twenty-three years in one of Oppolzer's cases).

Death from uræmia (vide page 258) occurs more frequently than in other forms of Bright's disease.

In other cases death is the result of the sudden development of inflammations of the serous membranes. Pericarditis and peritonitis are the most dangerous; recovery often occurs after pleurisy.

Pneumonia, pulmonary œdema, or œdema of the glottis may also prove rapidly fatal.

IV. DIAGNOSIS.—The diagnosis is easy if the urinary changes, cardiac hypertrophy, and retinal changes are present; sometimes a single one of these symptoms (generally the urinary changes) will suffice for diagnosis.

The disease is distinguished from acute nephritis and chronic parenchymatous nephritis by the large amount of urine, diminished specific gravity, small amount of albumin, scanty sediment, absence of blood in the urine, frequent absence of œdema. When an acute exacerbation occurs in chronic interstitial nephritis, the urine becomes bloody and scanty and the specific gravity increases, but the further course of the disease will clear up the diagnosis. The urine may become small in amount and the specific gravity may increase if the vigor of the heart's action fails, but these symptoms disappear as soon as the power of the heart is restored.

The disease is distinguished from waxy kidneys by the etiology, cardiac hypertrophy, retinal changes, and the absence of waxy liver and spleen.

With regard to the differentiation of primary and secondary cirrhosis of the kidneys we refer to page 280.

V. PROGNOSIS.—Recovery in this disease is impossible.

VI. TREATMENT.—The treatment is similar, in general, to that of acute and chronic parenchymatous nephritis. In addition, mental and bodily rest, milk diet, avoidance of strong tea and coffee, etc. (vide page 246).

In other cases we must adopt causal treatment; for example, in gout, syphilis, intermittent fever, wounds, and suppurations.

Iodide of potassium is recommended by many, but does not possess any special advantages.

5. *Suppurative Nephritis.*

(Abscess of the Kidney.)

I. ETIOLOGY.—Bacteria play a prominent part in the etiology of this disease. It is distinguished from Bright's disease by the tendency to the formation of abscesses and by the fact that it begins with small, circumscribed foci of inflammation. The bacteria may be carried to the kidneys through the blood-vessels or the urinary passages.

In acute septic endocarditis, the renal vessels may be filled with plugs of bacteria, which have been conveyed from the endocardium. Similar conditions may obtain in the suppurative nephritis which sometimes develops during septicæmic, pyæmic, and puerperal processes. In all

probability this is also true of various infectious diseases (osteomyelitis, typhoid fever, diphtheria, dysentery, phthisis, actinomycosis). Boettcher observed suppurative nephritis following abscess of the lung, and found elastic fibres of the lung in the abscess within the kidney, thus proving its embolic origin.

The bacteria are conveyed along the urinary passages in those cases in which inflammatory changes in these parts precede the renal disease. This is seen in pyelitis, occlusion of a ureter with subsequent stasis of urine and inflammation, cystitis, paralysis of the bladder in spinal diseases with subsequent decomposition of urine and cystitis, hypertrophy of the prostate, urethral stricture, etc.

But the inflammation does not always spread directly from the urinary passages to the kidneys. The foci of inflammation in the kidney and in the urinary passages are sometimes separated from one another by a long stretch of healthy tissue, so that it must be assumed that the bacteria are conveyed to the kidneys through the blood-vessels or perhaps the lymphatics. Suppurative nephritis is sometimes observed after trifling surgical operations on the genito-urinary apparatus; for example, after catheterization (surgical kidney).

The renal inflammation is sometimes propagated from adjacent parts (paranephritis, perinephritis; cold abscess in vertebral caries; hepatic, splenic, psoas abscess, etc.). It is sometimes the result of injury.

In a number of cases no cause can be discovered (idiopathic suppurative nephritis).

II. ANATOMICAL CHANGES.—These depend in part upon the etiology in each individual case. If the disease is the result of septic endocarditis or pyæmic and septicæmic processes, both kidneys are affected. The size of the organ is often unchanged. The capsule is more firmly adherent in places, and the otherwise smooth surface is slightly cloudy in such places. On section the kidney is found infiltrated with more or less numerous small, grayish-white foci which are often surrounded by an hyperæmic zone. In the cortex they are usually round, in the medullary substance they follow the direction of the tubes. The changes are especially pronounced in the papillæ.

Numerous blood-vessels in the kidneys are filled with emboli of bacteria; in the cortex, this is observed particularly in the loops of the Malpighian bodies. Immediately around the emboli are more or less numerous round cells, which increase in number and form miliary abscesses. The emboli also affect the epithelium of the tubes; the cells become swollen, lose their nuclei, and undergo coagulation-necrosis. The tubules themselves are not infrequently occluded by masses of bacteria and are dilated in places. Litten reports a case in which this gave rise to anuria and uræmia.

If suppurative nephritis has followed inflammation of the urinary passages, it is generally unilateral. The inflammation can usually be traced from the pelvis of the kidney to the papillæ. The foci of inflammation in the kidneys always contain bacteria. In this form of the disease miliary abscesses first develop, but they coalesce and finally convert the organ into a sac of pus, which can sometimes be felt during life beyond the median line of the abdomen.

Renal abscesses sometimes perforate into the pelvis of the kidney, peritoneal cavity, colon, small intestines, stomach, retroperitoneal cellular tissue, liver, through the diaphragm and lungs into the bronchi, or externally through the abdominal walls.

The pus is sometimes converted gradually into a chalky mass, which contains cholestearin crystals, phosphate and carbonate of lime, and granular detritus. Or the pus disappears more and more, and is replaced by fibrous cicatricial tissue, retraction and deformity of the kidney.

In traumatic cases very extensive changes are usually present, and extravasations and inflammations are generally found in the vicinity of the kidney. The greater part of a kidney is occasionally converted into a pulpy, purulent mass.

III. SYMPTOMS AND DIAGNOSIS.—The diagnosis is often impossible, especially in metastatic nephritis, in which the foci may be very numerous, but so small that they produce no notable changes.

If the disease has been preceded by injury, the patients usually complain of pain in the region of the kidneys, often radiating towards the umbilicus, thighs, and scrotum. They generally lie persistently on the healthy side in order to avoid an increase of the pain. We may notice a distinct chill, or repeated chilly sensations, followed by high fever and its associated symptoms. Diuresis, in the beginning, is sometimes entirely abolished; hæmaturia is often noticed at a later period. We may not conclude that the injury has given rise to suppuration unless masses of pus appear in the urine, or suppuration occurs in open wounds of the kidney. Some cases are very quickly fatal, others last for many weeks and months.

In non-traumatic cases the diagnosis is often much more difficult. The symptoms of the primary disease are sometimes so prominent that those of the secondary nephritis are entirely overlooked. In other cases the symptoms are those of internal suppuration: repeated chills, a remittent fever, profuse sweats, diarrhoea and increasing emaciation; perhaps sudden signs of perforation-peritonitis, which is found, on autopsy, to have resulted from a ruptured renal abscess.

The diagnosis may be regarded as positive if we can demonstrate a fluctuating renal tumor, a purulent urinary sediment, the passage of pieces of kidney through the urine, or rupture of the pus externally. It is still more certain if several of these symptoms are present.

A fluctuating renal tumor is not often observed in suppurative nephritis, since the abscess must attain considerable dimensions to permit of its being felt through the abdominal walls. In a few cases the tumor has extended to the median line. It is distinguished from tumors of the liver and spleen by the absence of respiratory displacement, and from perinephritic and paranephritic changes by the fact that it is more distinctly circumscribed. It must be remembered, however, that echinococci and soft tumors of the kidneys may also present fluctuation.

Pus will not appear in the urine, in suppurative nephritis, until the abscess has entered the pelvis of the kidney. The pus sometimes forms a sediment amounting to one-fourth the volume of the urine. Since it may also be derived from suppurative inflammation of the pelvis of the kidneys or the lower urinary passages, the diagnosis of nephritis may be very difficult, especially if local renal changes are absent. Mistakes may arise, even if a tumor is demonstrable in the renal region. Ogle reports a case in which a large abscess, which started from the tubercular second lumbar vertebra, was situated behind the left kidney. The pus passed down to the left side of the bladder, into which it ruptured.

If the urine contains pus, it is very apt to undergo decomposition. In determining whether suppurative nephritis is present, in addition to

suppurative inflammation of the lower urinary passages, we should ascertain whether the urine contains more albumin than corresponds to the amount of pus, and whether it contains casts, since both conditions indicate, though not indubitably, an implication of the kidneys.

The discharge of pieces of kidney in the urine would be a very valuable diagnostic sign, but it occurs with extreme rarity. Elastic fibres and shreds of connective tissue are found in the urinary sediment of renal tuberculosis, but tubercle bacilli are also present under such circumstances.

Rupture of the pus externally sometimes leads us on the right track. If the perforation takes place through the integument, the escaping pus is often mixed with urine. In perforation into the intestines, the stools are purulent and sometimes contain constituents of the urine; in one case Ogle found a renal calculus in the fæces. The pus may also rupture into the stomach, bronchi, retroperitoneal cellular tissue, or peritoneal cavity.

All the other symptoms are still more vague than those previously mentioned. We may mention pain in the kidneys, strangury, fever, etc.

The disease may last for months. Death will occur in a short time if the other kidney has been previously diseased. The fatal event is the result of exhaustion, occasionally of uræmia or perforation.

The disease is sometimes complicated by paralysis of both legs (affection of the spinal cord as the result of ascending neuritis).

IV. PROGNOSIS.—The prognosis is always grave, although recovery takes place occasionally despite advanced changes. In some cases the prognosis is unfavorable on account of the primary disease.

V. TREATMENT.—In many cases the treatment must be prophylactic and causal (avoidance of catheterization with unclean instruments, treatment of strictures and hypertrophy of the prostate, treatment of vesical and renal calculi, etc.). If the disease is the result of injury, we should order absolute rest, and the application of an ice-bag. If the existence of a renal abscess has been determined with certainty, reliance must be placed chiefly on surgical measures (puncture, aspiration, nephrotomy). In other respects the treatment is purely symptomatic.

6. *Paranephritis.*

I. ETIOLOGY.—The term paranephritis refers to inflammatory changes in the loose connective tissue around the kidney. This is connected directly with the cellular tissue of the pelvis, so that inflammatory processes often spread from one region to the other.

The disease is almost always acute; in rare cases it is chronic from the beginning. The noxious causes sometimes act locally, sometimes they are general in their nature. Moreover, we may distinguish between primary and secondary paranephritis.

Primary paranephritis is rarer than the secondary form; its chief causes are cold and injury (fall, blow, lifting heavy weights, etc.).

Secondary paranephritis includes those cases which are propagated from adjacent parts or follow certain infectious diseases.

Paranephritis is observed most frequently after pyelitis and pyelonephritis, much more rarely after renal abscess, tuberculosis, cancer, cysts, echinococcus, infarction, gangrene, or strongylus gigas of the pelvis of the kidney. In some

cases it follows primary chronic cystitis or urethral stricture, the inflammation being conveyed either along the mucous membrane or the surrounding connective tissue.

Inflammation of the pelvic cellular tissue spreads not infrequently to the tissue around the kidney. Paranephritis is occasionally secondary to peritonitis, cold abscess of vertebral caries, psoriasis, hepatic and splenic abscess, typhlitis, etc. In rare cases it is secondary to diseases of the thoracic organs. For example, Bayer observed it after perforation of a pulmonary cavity through the diaphragm.

Among the infectious diseases which may act as a cause of paranephritis, we may mention small-pox, typhoid and typhus fevers, pyæmia, and puerperal fever.

In a certain proportion of cases no cause can be discovered. The disease is more frequent in men than in women, and generally develops from the twentieth to sixtieth years; it is not infrequent in childhood.

II. ANATOMICAL CHANGES.—Paranephritis is almost always unilateral. Sometimes we find a diffuse purulent or sero-purulent infiltration of the paranephritic connective tissue, sometimes a circumscribed, encapsulated abscess, which often contains shreds of connective tissue. The pus sometimes has a fæcal odor, although it does not communicate with the intestines. The fat in the tissue has often disappeared entirely. The surface of the kidney may present losses of substance and sometimes there is considerable destruction of the parenchyma. The kidney may float to and fro in the surrounding mass of pus.

In rare cases gangrenous changes are noticed, much more frequently perforation ensues. If the perforation takes place into the peritoneal cavity, rapidly fatal peritonitis results. The pus often undermines the muscles of the loins and finally appears under the integument. As we have previously stated, the pus may also burst into the pleural cavity, the lungs and bronchi, the colon, stomach, or duodenum, or it may follow a sinuous course and appear beneath the integument near Poupart's ligament, in the raphe of the scrotum or perineum. Perforation has also been observed into the vagina, bladder, urethra, hip joint. In one of my cases the vertebræ were eroded.

In rare cases the pus does not perforate, but is entirely absorbed; caseation of the pus may also develop. Cicatricial fibrous tissue sometimes forms and, by its retraction, may cause atrophy of the kidney. In a case reported by Elias, this lesion gave rise to fatal stenosis of the portal vein.

Pleuritis on the same side as the paranephritis is a not infrequent complication; in rarer cases, pericarditis develops. Amyloid degeneration of the kidneys ensued in one of my cases which had lasted more than ten months.

III. SYMPTOMS.—In secondary paranephritis, the symptoms often develop very insidiously. In the primary form, the initial symptoms are usually violent. The disease begins with one or more chills, followed by high fever which is either continued or markedly remittent. Then follow violent pains in the region of the kidneys, complete anorexia, increased thirst, frequently vomiting.

Fever, pain, and swelling are the cardinal symptoms.

Febrile movement is rarely absent. Occasionally it presents a distinct intermittent type, but it is usually remittent. If perforation of pus occurs suddenly or an incision is made into the abscess, the fever sometimes ceases abruptly and does not return until retention of pus takes place. In very acute cases, typhoid symptoms may make their appearance.

Pain is also a very important symptom. In the majority of cases, it is confined to the renal region, sometimes upon the affected side, sometimes extending to the healthy side. It is occasionally constant, in other cases intermittent; it increases on pressure. In rare cases it radiates into the genitals or thighs; numbness and weakness of the thighs have also been observed.

A tumor is not infrequently visible externally, especially in the erect position. The integument over the region of the kidneys is destitute of folds, shining, œdematous, not infrequently very red and hot. If one hand is placed upon the renal region, and the other hand is pressed towards it from the anterior abdominal walls, we will detect, at first, only a diffuse resistance and infiltration, later a circumscribed tumor.

As a rule, the patients lie upon the back, and slightly turned towards the affected side. The spine is curved, with its concavity directed towards the lesion, the hip and knee joints are usually flexed, and the thigh is often rotated outwards. The patients avoid changing the position of the body, since the slightest tension of the abdominal walls is attended with pain.

Constipation is often the result of compression of the ascending or descending colon by the tumor. Dyspnœa is the result of interference with the movements of the diaphragm.

The urine presents no special changes.

In some cases, the disease runs its course in two to four weeks; in others, it lasts for months, and finally proves fatal by exhaustion.

When rupture of the pus externally is impending, the skin becomes redder and more prominent, grows thinner, and finally ruptures. A fecal odor of the pus does not necessarily indicate the existence of a communication with the intestine. The pus sometimes has a putrid odor and contains gangrenous shreds of tissue. Cutaneous emphysema was observed by Trousseau in two cases. Perforation may also take place into the intestine, stomach, peritoneal cavity, pleural cavity (pyo-pneumothorax will develop if the intestine and pleural cavity are perforated at the same time), lungs and bronchial tubes, urinary passages.

Rupture into the peritoneal cavity is the most dangerous. Perforation into the lungs may also be followed by death from suffocation. Rupture into the intestines is by no means unfavorable, since feces rarely enter the abscess cavity and give rise to gangrene, on account of the usually valve-like structure of the fistula.

In rare cases dangerous hemorrhage takes place into the abscess cavity.

IV. DIAGNOSIS.—The disease may be mistaken for

a. Lumbago. The pain is more superficial, fever and swelling are absent.

b. Cutaneous abscess in the loins. The symptoms are milder and more superficial; palpation anteriorly gives negative results.

c. Empyema necessitatis, which opens in the lumbar region. We must take into consideration the development of the disease, the absence of pain over the region of the kidneys, the absence of a tumor and of the characteristic position of the body.

d. Abscess in vertebral caries. The renal region is free from pain, while the spinous processes of the vertebræ are usually sensitive.

e. Diseases of the kidney or the pelvis of the kidney (tuberculosis, cancer, abscess, etc.). One or the other of the cardinal symptoms will be absent. In addition, paranephritis extends posteriorly, while the other conditions mentioned have a tendency to spread anteriorly.

f. Typhlitis, paratyphlitis, and perityphlitis. The pain and swelling are situated deeper, gastro-intestinal symptoms are prominent, and the amount of indican in the urine is increased.

g. Psoitis. The localization of the pain is different; in paranephritis, moreover, the flexed leg can usually be extended without pain; this is not true of psoitis.

h. Coxitis. Movements of the leg, especially rotation, are very painful.

i. Other abdominal tumors. The symptoms to be considered in the discussion of renal cancer will hold good here.

V. PROGNOSIS.—This depends chiefly upon the primary disease. It is rendered worse by the occurrence of perforation, and is always bad in rupture into the peritoneal cavity.

VI. TREATMENT.—The treatment is purely surgical. Poultices should be applied and, when fluctuation occurs, a free incision should be made. Spontaneous absorption can be looked for in exceptional cases alone. If the pain is very severe, subcutaneous injections of morphine may be made. In addition, the bowels should be kept open.

APPENDIX.

Perinephritis is an inflammation of the fibrous capsule of the kidney. Chronic perinephritis gives rise to thickenings and opacities of the capsule, and adhesions to the surface of the kidney. It often accompanies various renal diseases. This is also true of acute perinephritis. The latter sometimes results in the formation of foci of pus, between the capsule and the surface of the kidney.

7. Embolism and Hemorrhagic Infarction of the Kidneys.

I. ETIOLOGY AND ANATOMICAL CHANGES.—Renal embolism is occlusion of the trunk or branches of the renal artery by masses which have been conveyed thither by the current of blood. The emboli may act in a mechanical or specific manner. The latter form has been discussed under the head of suppurative nephritis.

Emboli which act in a mechanical manner take their origin, as a rule, in the left heart, usually from vegetations of the mitral or aortic valves. They are derived less frequently from atheromatous changes in the aorta.

The left renal artery is more frequently the site of embolism than the artery of the right side, because it is given off at a less acute angle from the aorta. Several emboli are often found in one kidney.

If the main trunk of the artery is occluded, the entire kidney may undergo necrosis. Embolism of smaller branches is usually followed by the formation of infarctions (white and red). Both are wedge-shaped, with the broad base directed towards the surface of the kidney.

The white infarction forms a wedge-shaped area of anæmic, necrotic tissue. The epithelium cells of the tubes are converted into non-nucleated, shining masses (coagulation-necrosis). As a rule, the periphery is surrounded by an hemorrhagic zone. This is the more frequent form of infarction.

The hemorrhagic infarction is a wedge-shaped, hemorrhagic area, of a dark, blackish-red color, and granular surface. At a later period the infarction becomes discolored, the centre being first converted into a gray, grayish-yellow, or yellow mass. The cellular elements may undergo fatty degeneration and absorption, leaving a depressed cicatrix.

Embolism is not the sole cause of hemorrhagic infarction. This is

produced when the circulation is interrupted from any cause, such as injury and rupture of the renal artery.

II. SYMPTOMS AND DIAGNOSIS.—In the majority of cases, perhaps, renal embolism remains latent during life. The disease may be suspected if a patient suffering from valvular disease of the heart is suddenly seized with pain and tenderness in the region of the kidneys, vomiting, chill, high fever, and hæmaturia. In addition, the urine contains albumin and casts.

III. PROGNOSIS AND TREATMENT.—The prognosis depends on the primary affection. The treatment is symptomatic.

8. Waxy Degeneration of the Kidneys.

I. ETIOLOGY.—The causes are the same as those of waxy degeneration of other organs (vide page 213). According to Bartels, suppuration is especially apt to give rise to waxy changes when it causes molecular degeneration of the tissues and communicates with the external air. The lesion may develop within three months from the beginning of suppuration. Bull claims to have observed waxy degeneration of the kidneys on the eighteenth day of an acute psoas abscess. Suppuration in one kidney (pyelonephritis or paranephritis) sometimes causes waxy degeneration of the other kidney.

In rare cases no cause is discoverable. The disease is more frequent in men than in women, and from the tenth to fiftieth years. It may develop in children as the result of congenital syphilis.

II. ANATOMICAL CHANGES.—Since the causes of waxy kidneys may also give rise to Bright's disease, the former are usually associated with chronic parenchymatous or interstitial nephritis. Both kidneys are almost always affected, though sometimes in different degrees. Wilson recently described waxy degeneration of one kidney; the other had shrunk to a body which looked like a lymphatic gland.

The first stages of the disease can only be recognized with the microscope or by the aid of suitable reagents (vide page 214). In more advanced stages, the affected glomeruli are recognized as gray, slightly transparent dots, which assume a mahogany color when treated with iodine.

When the degeneration is extensive and diffused, the kidney is increased in size (sometimes more than double). The capsule is readily removed; the surface is smooth and very pale, in places a few congested stars of Verheyen are noticeable. On section, the organ is extremely pale and has a waxy color; it is peculiarly hard and brittle. The cortex appears to be affected chiefly or exclusively. The waxy color is sometimes confined to this portion, while the medullary substance may be congested. On the application of iodine, we notice reddish-brown dots (glomeruli) and streaks (affected blood-vessels). In one case of pure waxy kidneys, Rindfleisch found that the bases of the pyramids were separated from one another by furrows which were as deep as in the foetal kidney.

The loops of vessels within the Malpighian bodies are first affected. They assume a peculiar dull lustre, become thickened, lose their fibrous structure, and if all the loops are affected, the glomerulus increases not inconsiderably in size. The more the waxy degeneration increases, the more the nuclei and epithelium are destroyed. Then the vasa afferentia and vasa recta are affected, later the intertubular capillaries. The capillaries of the medullary portion are affected only in

more advanced cases. Bowmann's capsule and the tunica propria of the tubes may also be affected, and converted into shining, swollen structures which narrow the lumen of the tubes. Eberth found that the walls of the straight tubes are affected very early and to a marked degree. The epithelium cells may also undergo waxy degeneration, especially in the papillæ (Kyber). These are sometimes found in the urinary sediment. The tubes not infrequently contain casts, which are sometimes the result of agglutination of degenerated epithelium cells.

In rare cases the kidney contains large collections of amyloid substance which appear macroscopically as gray patches. Waxy degeneration has also been observed in the walls of the main trunk of the renal artery. Thrombi have been found in the renal vein.

The swelling of the walls of the vessels may be so great as to prevent artificial injection of the renal artery, though this does not always happen.

Cases of pure waxy degeneration of the kidneys are rather exceptional. As a rule, epithelial and interstitial changes are also present, and, indeed, the latter usually predominate. But we must not look upon every fatty degeneration of the epithelium cells of the tubes, or every accumulation of granular cells and drops of fat in the lumen of the tubes as products of inflammation, since the diminution of the blood supply following the narrowing of the vessels may give rise to purely degenerative processes in the epithelium cells. It is often difficult to determine whether we have to deal with inflammation or simple degeneration.

Waxy degeneration is also found, in almost every case, in the liver, spleen and intestines; often in many other organs. According to some authors, waxy degeneration of the kidneys never occurs alone. Among seventy-six cases collected by Rosenstein, the kidneys were alone affected in five cases.

III. SYMPTOMS.—Since pure waxy kidneys are so extremely rare, it is readily understood that the symptoms sometimes approach those of chronic parenchymatous nephritis, sometimes those of interstitial nephritis.

In rare cases extensive amyloidosis of the kidneys may exist without albuminuria. Anasarca may be present or absent; in the former event it is usually the result of cachexia.

As a rule, however, the urine presents very marked changes.

The amount of urine is generally below the normal, but presents numerous variations. The reaction is acid, the specific gravity varies from 1.010 to 1.015. As a rule, it contains very little or no sediment. In the latter are found narrow hyaline casts, which are not infrequently covered with drops of fat, round cells, or epithelium from the tubes, occasionally with fatty epithelium, rarely with a few round cells which present an amyloid appearance. The casts sometimes have a waxy gloss, are unusually broad and fissured, often jagged, and sometimes present a waxy reaction. Red blood-globules are rarely present. The urine contains albumin, the daily amount of which may exceed twenty grams.

The amount of urea and sodium chloride is unchanged; that of phosphoric acid is diminished. The percentage of indican is very much increased. In one case, in which the suprarenal capsule was also very waxy, Virchow found an unusual amount of pigment in the urine.

Anasarca is an almost constant symptom. Dropsy of the serous cavities occurs less frequently.

As a rule, the skin is pale; according to Grainger Stewart, pigment is deposited in the face, especially the eyelids, and the veins of the

cheeks become dilated. Emaciation does not occur in all cases. The bodily temperature and the pulse are usually unchanged.

Changes in the circulatory apparatus are rarely observed; hypertrophy of the left ventricle is almost always absent.

Diseases of the respiratory apparatus, particularly phthisis, often form the starting-point of the renal disease. It is said that the pulmonary changes not infrequently cease to advance after the development of the renal affection.

Changes in the digestive tract are often present, particularly intestinal ulcerations and very profuse diarrhœa, which are sometimes a cause, sometimes a complication of the kidney lesion. Retinal changes develop very rarely.

Death results from uræmia in extremely rare cases. Inflammations of the serous membranes (peritonitis, pleuritis, pericarditis, meningitis) are sometimes the cause of death. The fatal termination may also follow an acute pneumonitis. In the majority of cases, death is the result of marasmus, which is complicated not infrequently by marantic thrombosis of one of the veins of the leg. Diuresis is often very much diminished towards the close of life, on account of the failing power of the heart.

In very rare cases the disease terminates in recovery.

The duration of the disease varies greatly; in some cases it lasts more than ten years.

If waxy kidney is complicated with chronic parenchymatous or interstitial nephritis, the symptoms will approximate those of the latter disease.

IV. DIAGNOSIS.—The diagnosis is impossible if albuminuria is absent. If this symptom is present, we should take into consideration the character of the urine, the etiology, and the evidences of waxy degeneration of the liver and spleen. It is distinguished from acute diffuse nephritis by the fact that hæmaturia is almost always absent, the specific gravity is lower, and the constituents of the sediment are entirely different.

In chronic parenchymatous nephritis, the urine is more scanty and darker, the specific gravity is higher and the sediment more abundant.

In chronic interstitial nephritis the amount of urine is increased, the specific gravity diminished, the amount of albumin small; we also find cardiac hypertrophy, retinal changes, and absence of anasarca.

In the mixed forms of nephritis, waxy kidneys can only be diagnosed if we are able to demonstrate waxy changes in the liver and spleen.

V. PROGNOSIS.—This is generally unfavorable on account of the primary disease. According to Gerhardt, recovery is not very rare in childhood.

VI. TREATMENT.—In some cases the prophylaxis consists of surgical operations (opening of abscesses, etc.). Causal treatment must be instituted in syphilis, intermittent fever, etc.

In other cases, we must rely on nutritious diet, and preparations of iron, iodine, and iodide of iron. Budd and Rosenstein recommend the administration of nitric acid.

9. Cloudy Swelling of the Kidney and Fatty Kidney.

1. Fatty kidney may result from the introduction of fat into the kidneys through the medium of the blood (fatty infiltration) or conversion of the renal parenchyma into fatty substances.

An abnormal accumulation of fat in the organ is found in very puffy and fat individuals.

Fatty degeneration is not infrequently a result of previous inflammation, but we will discuss only those forms which develop without a prior inflammatory stage.

The causes are the same as those of fatty degeneration of the liver, viz., anæmic and cachectic conditions (phthisis, cancer, scrofula, protracted suppuration, profuse hemorrhages, etc.). This category also includes the fatty kidney of old age.

The disease is not infrequent in febrile infectious diseases (typhoid and typhus fevers, pyæmia, etc.), and it has also been observed after extensive cutaneous burns.

Another group of cases is the result of toxic agents, such as phosphorus, arsenic, carbonic oxide, etc.

In certain cases it is the result of local circulatory disturbances, such as occur, for example, in embolic processes.

2. As in the case of the liver, fatty degeneration and infiltration of the kidneys cannot be distinguished from one another by the aid of the microscope.

Fatty degeneration is preceded by a stage of cloudy swelling (parenchymatous degeneration). Both processes affect the cells of the convoluted tubes, while the Malpighian bodies and other parts are unaffected. In the condition of cloudy swelling, the epithelium cells have a peculiar coarsely granular, opaque appearance, but clear up on the addition of acetic acid or potash. Later minute drops of fat make their appearance; these are not dissolved in acetic acid and potash, but are soluble in alcohol and ether. Some of the cells are entirely converted into granulo-fatty cells and may undergo dissolution. This condition may undergo resolution, a part of the fat being removed by the urine, the rest being absorbed.

Fatty kidney is recognized macroscopically by the light, butter-yellow color, flabby consistence, and the discharge, on pressure, of a fatty fluid.

In addition to the kidneys, the liver is almost always fatty, and not infrequently the heart. At the same time the glandular epithelium of the gastric mucous membrane generally undergoes fatty degeneration.

3. In the majority of cases the condition cannot be recognized during life. In rare cases the urine contains a visible amount of fat. Slight albuminuria is also noticeable in a few cases.

4. The prognosis and treatment depend on the primary disease.

10. *Cancer of the Kidney.*

I. ETIOLOGY.—Renal cancer is primary or secondary. Secondary renal cancer develops through the agency of the blood-vessels or spreads directly from adjacent parts. It is not infrequently secondary to cancer of the testicles, and sometimes appears years after the extirpation of the latter.

In the majority of cases of primary renal cancer no cause can be ascertained. Traumatism is sometimes mentioned as an etiological factor.

Congenital cancer of the kidneys has been observed in a few cases.

The disease is more frequent in men than in women. It is most common during the first five years of life and beyond the age of fifty years.

II. ANATOMICAL CHANGES.—As a rule, secondary renal cancer affects both kidneys, the primary form is unilateral; the latter develops more frequently in the right kidney.

Secondary cancer generally appears in the form of circumscribed nodules, primary cancer in the form of a diffuse infiltration.

Almost all varieties of cancer are observed in the kidney (usually medullary, more rarely alveolar cancer). Cancer and sarcoma are sometimes associated; Wagner reported a case of primary cylindrical cancer.

The kidney, as a rule, is increased in size and sometimes exceeds the dimensions of a man's head. Its shape is often very little changed. Upon section of the organ we find either circumscribed cancerous

nodules or a diffuse infiltration. The nodules sometimes possess a sort of fibrous capsule. The lesion always begins in the cortex and often is confined to this part. The consistence varies according to the predominance of cancer stroma or cells. The tumors are white or rosy-red (abundance of vessels). Rupture of vessels and extravasations are sometimes produced. If the hemorrhages have lasted for some time, we will find cavities filled with a chocolate-colored fluid (red blood-globules, fatty cancer cells, fat crystals, and detritus).

According to Waldeyer, the cancer cells are the result of a proliferation of the epithelium cells of the convoluted tubes.

The unaffected kidney may be merely hyperplastic or in a condition of waxy degeneration. Badt and Rosenstein reported a case of cancer of one kidney, and tuberculosis of the other.

The increased size of the organ may cause displacement of the spleen, liver, stomach, and intestines. Gastroectasia has been observed in cancer of the right kidney as the result of stenosis of the duodenum.

The growth may proliferate into the pelvis of the kidneys and the ureters; particles may break off, occlude the ureters, and give rise to hydronephrosis. The pelvis of the kidney is often filled with coagula of blood. According to Kuehn, the pelvis of the healthy kidney may contain blood if the organ is excessively congested.

The cancer extends occasionally to the renal veins and sometimes to the inferior vena cava (in one case to the right heart). It may give rise to embolism of the pulmonary artery, and sudden death. Cases have also been reported in which the cancer penetrated the iliac and azygos veins.

The adjacent lymphatic glands are often involved and may compress the kidneys, blood-vessels, and the ureters. In rare cases the cancer ruptures into the peritoneal cavity, the intestines, or externally through the abdominal walls.

Metastases occur very rarely in primary renal cancer; they have been noticed most frequently in the lungs. Gerstaecker observed a rare case of metastasis in the muscles.

III. SYMPTOMS.—Many cases are unrecognized during life. This is particularly true of secondary cancer, because the primary cancer in other organs attracts the chief attention.

Sometimes the sole symptoms are increasing cachexia and marasmus. In other cases, there are obstinate nervous symptoms (neuralgia of the lower intercostal nerves, ileo-lumbar neuralgia, sciatica, formication, and numbness in one leg, diminished muscular power and emaciation), and at the autopsy they are found to be the result of compression of the nerves by a latent renal cancer.

In a third series of cases, unexpected hæmaturia develops and may prove fatal within a single day.

The positive diagnosis of cancer of the kidney depends on the demonstration of a renal tumor and the occurrence of hæmaturia.

In some cases, the tumor is so large as to distend the abdomen to a considerable extent. It is characteristic of these tumors that, as a rule, they are traversed by the ascending colon (on the right side), or the descending colon (on the left side). Peristaltic movements or temporary tympanites is sometimes observed in those parts of the colon which are situated in front of the tumor. The colon is occasionally flattened to

such an extent that it is no longer visible, but can be felt with the hand. Or gentle percussion over it gives rise to a tympanitic sound, which gives place, on firm percussion, to the dull sound of the underlying tumor. In doubtful cases, the colon may be distended, through the anus, with gas in order to render it visible. Superiorly, the tumor is separated from the liver (or spleen) by a tympanitic zone (the transverse colon).

It undergoes no respiratory displacement and, as a rule, is altogether immovable, unless a floating kidney has been affected. It is usually nodular, tender on pressure, occasionally distinctly fluctuating in places. In the latter event puncture has sometimes been performed; the fluid was either bloody, chocolate-colored, and contained urea and uric acid, or it contained whitish particles of cancer tissue. Pulsation is sometimes transmitted to the tumor.

Hæmaturia is a very frequent but not a constant symptom, and may occur very early or shortly before death. The urine may be purely blood or merely bloody, sometimes contains clots of the shape of the ureter, and is occasionally fœtid. These clots may occlude the ureter or urethra, and give rise to colicky pains, diminution of the urine or anuria. Sudden occlusion of the ureter may also occasion a profuse hemorrhage. In rarer cases, the hemorrhage is derived from the relatively healthy, congested kidney.

Among 115 cases of primary renal cancer, Rohrer found :

Latent cancer without tumor and hæmaturia,	36 (31 per cent)	
Suspicious cases with hæmaturia alone,	12 (11	")
Exquisite cases with tumor and hæmaturia,	25 (22	")
Tumor alone,	42 (36	")
Demonstrable tumor,	47 (58	")
Hæmaturia,	37 (32	")

Occlusion of the urinary passages may also be produced by large particles of cancer. If these enter the urine, they form an extremely valuable diagnostic factor. The hemorrhages are unattended with pain, if the urinary passages are not occluded by clots.

When hæmaturia is not present, nothing abnormal is detected in the urine, unless the patient is suffering from some other renal disease.

Other symptoms are not very decisive. We may mention pain, which is either confined to the region of the kidneys, or radiates along various nerve tracts (intercostal and sciatic nerves), etc. Paræsthesia, anæsthesia, and paralysis of one leg are occasionally observed. Paraplegia may be produced if the cancer proliferates into the retro-peritoneal cellular tissue, perforates the vertebræ, and compresses the spinal cord.

The patient assumes a cachectic appearance. In some cases there is obstinate insomnia. Constipation is generally produced by compression of the intestines.

Icterus has been occasionally observed. Kuehn described, in one case, abnormal formation of hair and pigment.

The average duration of the disease is one year. Dunbop reported three cases in which the duration varied from ten to seventeen years. It rarely proves fatal within a couple of weeks from the appearance of the first symptoms.

Death sometimes occurs suddenly from hæmaturia or hemorrhage into the peritoneal cavity. In other cases it is the result of increasing marasmus, occasionally of peritonitis. Finally, the fatal termination

may be brought about by perforation internally or externally, with subsequent gangrene, in rare cases by uræmia.

IV. DIAGNOSIS.—Renal tumors may be mistaken for tumors of the liver, spleen, stomach, intestines, pancreas, lymphatic glands, for ovarian cysts, aneurisms, and psoas abscess.

In contradistinction to hepatic humors, those of the kidney present no respiratory nor passive mobility; hepatic dullness is separated from that of the renal tumor by a tympanitic zone; hepatic symptoms are usually absent; if the patient is lying on the back the hand can generally be inserted between the tumor and the ribs, but this cannot be done in tumors of the liver; renal tumors generally have the shape of the kidney, and the colon is situated on their anterior surface.

Tumors of the spleen also, as a rule, possess respiratory and passive mobility; the anterior edge of the spleen usually presents the characteristic notches. Moreover, splenic tumors grow upwards and push the left hypochondrium outwards, while tumors of the left kidney grow forwards and produce prominence of the anterior abdominal walls.

If there is a suspicion of tumor of the stomach, we should ascertain whether the tumor changes its position after distention of the stomach with carbonic acid gas.

Fæcal tumors can be excluded by the prolonged exhibition of cathartics.

Ovarian tumors grow from below upwards, and are in direct contact with the abdominal walls, while renal tumors are covered with loops of intestines; in ovarian tumors there are changes in the position of the uterus and disturbances of menstruation; finally, an exploratory puncture may be resorted to.

Psoas abscess is distinguished by the more acute pain and the peculiar position of the limb (flexion and adduction of the thigh).

In aneurism there is retardation of the pulse compared with the apex beat of the heart and pulsation of the tumor in all directions.

Renal cancer cannot be distinguished during life from cancer of the paranephritic connective tissue. It is distinguished from cysts, echinococcus, and abscess of the kidneys by the results of an exploratory puncture, from tuberculosis of the kidneys by the presence of tubercle bacilli in the urine.

If the diagnosis must be based on hæmaturia alone, the renal origin of the latter is recognized by the fact that the blood is mingled uniformly with the urine. The absence of casts, and a small amount of albumin (corresponding to the amount of blood) will serve to exclude nephritis.

The diagnosis is much easier if a renal tumor and hæmaturia are combined.

V. PROGNOSIS AND TREATMENT.—The prognosis is bad, as in all cancers. The treatment is purely symptomatic. Nephrotomy may be performed if one kidney is perfectly healthy.

APPENDIX.

Other varieties of neoplasm may develop in the kidneys. These produce either the same symptoms as cancer, or they are so small that they remain entirely latent during life.

a. Sarcoma of the kidneys is usually metastatic and bilateral. Primary sarcoma of the organ is extremely rare. Not infrequently we find mixed forms of sarcoma and carcinoma. Elber and Schueppel described a myxosarcoma of the kidney; three cases of myosarcoma striocellulare (a sarcoma with striated muscular fibres) have also been reported.

b. According to Sturm, adenoma occurs more frequently than is generally believed; it appears in the shape of small nodules or extensive tumors.

c. Fibromata generally appear as small nodules near the larger vessels at the periphery of the medullary substance. Wilks reports one case in which the entire right kidney was converted into a fibroma as large as a child's head.

d. Cavernomata are usually situated immediately beneath the surface of the kidney. They possess no clinical significance.

e. Lymphangiomata have the same significance as cavernomata. This is also true of myxomata, gliomata, and lipomata.

11. *Cystic Kidneys.*

a. Congenital Cystic Kidneys. Cystoid degeneration of the kidneys is sometimes congenital. One, or more frequently both, kidneys are converted into a multilocular structure, each chamber being filled with fluid contents. The number of cysts may be so large that hardly any normal renal tissue is left. At the same time, the organ increases in size to such an extent as to interfere with the delivery of the child. In other cases, the children are born alive, but soon die from interference with the movements of the diaphragm. Some of the children also present other malformations, such as hare-lip, club foot, etc. The mothers occasionally give birth to several children who suffer from cystic kidneys.

The chief part in the etiology seems to be played by mechanical obstructions in the urinary passages (inflammation of the renal papillæ, with subsequent obliteration, distention of the straight tubes with urates, congenital phimosis, etc.).

In certain cases the affection seems to be the result of a primary error of development. Kupfer showed that cystic kidneys may be associated with absence of the renal pelvis and ureter.

The cysts contain a thin, light-yellow, clear fluid, in which are found albumin, cholestearin, traces of uric acid (but no urea), carbonates and phosphates, occasionally colloid substances. The cyst-wall is composed of connective tissue, lined with endothelium cells.

In some cases congenital renal cysts appear to undergo enlargement in later years.

b. The formation of cysts in chronic interstitial nephritis has been previously discussed (Vol. II., page 282).

c. Renal cysts occasionally develop spontaneously, perhaps from the growth of fetal cysts. In other cases they are preceded by injury, and are probably the result of hemorrhage into the Malpighian corpuscles or tubes, with occlusion and subsequent dilatation. They are sometimes the result of occlusion of the ureters by calculi; the cysts themselves may contain several calculi.

The cystic change affects one or both kidneys. They are sometimes converted into a multilocular organ, in other cases more or less of the parenchyma of the kidney is retained. The organ often attains enormous dimensions, and may weigh as much as sixteen pounds. The individual cysts often contain septa; the contents may be thin and watery, colloid or hemorrhagic. They contain epithelium and round cells, red blood-globules, tablets of cholestearin, triple phosphates, fatty and granular detritus. Urea is generally absent, uric acid may also be absent. Tyrosin is often, leucin almost constantly, present. Litten described a case in which the mucous membrane of the ureters was also covered with numerous cysts. Hepatic cysts have also been found in a number of cases. Cardiac hypertrophy, especially of the left side, is present in many cases, even in unilateral cystic kidney.

The symptoms of extensive renal cysts are those of a fluctuating renal tumor. Changes in the urine may be absent; hæmaturia is sometimes noticed; albuminuria will be observed only when the kidney is otherwise diseased. The disease sometimes ends with uræmic symptoms. In other cases death is the result of suffocation from displacement of the

diaphragm, heart, and lungs. Suppuration of the cysts sometimes occurs, and is accompanied by chills, fever, and death from exhaustion. The cysts may also perforate into adjacent organs.

The diagnosis is very difficult; the tumor must be differentiated from renal abscess, cancer, tuberculosis, echinococcus, and hydronephrosis, and from ovarian tumors. Relative recovery sometimes follows puncture. The treatment in other respects is symptomatic.

12. *Echinococcus of the Kidneys.*

I. ETIOLOGY.—Echinococci develop much less frequently in the kidneys than in the liver or lungs. The disease is contracted, like echinococci of other organs, from the tænia of dogs (vide page 220). It occurs most frequently from the age of twenty to forty years, but has also been found in children of four years and in old people. It is somewhat more frequent in men than in women.

II. ANATOMICAL CHANGES.—As a rule, one kidney, generally the left, is alone affected. The anatomical appearances are the same as in echinococci of the liver. It is characteristic of renal echinococci that the fluid contents may contain crystals of uric acid, oxalate of lime, and ammonia magnesia phosphates. Hæmatoidin crystals are observed in a few cases.

Their size varies from that of a walnut to that of a man's head. As a rule, a certain part of the renal parenchyma is retained, and is often in a condition of fatty degeneration, interstitial connective-tissue proliferation, and atrophy. Extravasations of blood are often found around the vesicle. There is always only a single mother cyst, which sometimes adheres to adjacent organs by fibrous adhesions. As a rule, the starting-point is in the cortex.

Renal echinococci may undergo the same changes as those of the liver, *i. e.*, they may atrophy, remain stationary, enlarge and compress adjacent organs, suppurate and perforate. Perforation occurs most frequently into the pelvis of the kidney, and the vesicles, or their constituents, then appear in the urine. Perforation may also occur into the peritoneal cavity; in one case, the muscular tissue of the loins was perforated.

The unaffected kidney is usually hypertrophic.

III. SYMPTOMS.—The disease remains latent if a large tumor is not produced, or if perforation does not occur.

If a renal tumor alone is present, the disease may be mistaken for tuberculosis, cancer or cysts of the kidney, or hydronephrosis. Fluctuation and hydatid tremor may be absent, but may be present in other, even firm tumors. Exploratory puncture is often alone decisive; the fluid obtained is non-albuminous, and contains succinic acid, echinococcus hooklets, occasionally scolices.

Perforation into the pelvis of the kidney and evacuation of vesicles in the urine are generally attended with violent pains and the symptoms of renal colic. In some cases the evacuation of the vesicles is preceded for several days by pains in the region of the kidneys. It sometimes takes place spontaneously, in other cases it is preceded by a fall or blow, etc. According to some writers, evacuation of the vesicles is furthered by drinking strong coffee or tea. The patients sometimes experience a sensation as if something had burst within the abdomen. Violent pains may result from occlusion of the ureters by the echinococcus vesicles. This is attended in some patients with chill, fever, vomiting, and syncope. The pains are located at first in one loin, and then radiate into the mons veneris, testicle, or thigh; they sometimes cease abruptly as soon as the vesicle enters the bladder. Pain is again produced during the passage of the vesicles through the urethra. Intact vesicles, or portions of the echinococcus heads and hooklets, are found in the urine. Hæmaturia is not infrequent.

The renal tumor sometimes increases rapidly in size, so long as the vesicles occlude the ureter, and thus produce acute hydronephrosis.

The number of vesicles in the urine may vary from a few to fifty or more. They may continue to be evacuated for days, weeks, and months. Relapses sometimes occur at the end of several years.

In perforation into the stomach, vesicles are found in the vomited matters; in perforation into the intestines, they are found in the fæces. In perforation into the air passages, the vesicles are expectorated, and the sputum has a urinous odor. Occlusion of the bronchi may result in symptoms of suffocation.

Spontaneous recovery ensues not infrequently, especially after evacuation in

the urine. Danger may arise from suppuration of the echinococcus and compression of adjacent organs. Death has also been known to follow rupture of the kidney. The disease may last many years.

IV. DIAGNOSIS.—The diagnosis is very difficult, but is facilitated by an exploratory puncture. Evacuation of vesicles in the urine does not justify a diagnosis of echinococcus of the kidney, since the vesicles may have perforated the urinary passages from adjacent parts.

V. PROGNOSIS AND TREATMENT.—Spontaneous recovery is not infrequent. The parasite can be removed only by means of an operation (electrolysis, puncture, injection of iodine, cauterization, incision).

If the evacuation of the vesicles is attended with pain, we may administer narcotics, and facilitate their discharge by the abundant ingestion of warm tea, or gentle stroking of the affected ureter.

APPENDIX.

Pentastomum denticulatum and *cysticercus cellulosæ* have also been found in the kidney, but they possess no clinical interest.

13. *Floating Kidney.*

(*Movable Kidney. Ren migrans.*)

1. Floating kidney is the term applied to that condition in which the kidney undergoes active and passive movements in the abdominal cavity. Women are affected more frequently than men. As a rule, the condition develops between the twenty-fifth and fortieth years, but has also been observed in childhood.

In rare cases injury acts as the immediate cause. In others it is the result of increased weight of the kidney, caused by cancer, cysts, etc., of the organ. It has also been attributed to hard work, lifting heavy weights, obstinate vomiting, straining at stool, obstinate cough, tight corsets, pregnancy. In a few cases under my observation the patients were hysterical females, who also suffered from menstrual disturbances.

In some cases the conditions necessary to the development of floating kidney (absence of fat in the capsule of the kidney, loose and long folds of the peritoneum) seem to be congenital.

In the majority of cases the right kidney is affected, rarely the left kidney, still more rarely both kidneys. Even under normal conditions the right kidney is more movable than the left. This is owing to the respiratory movements of the liver. Moreover, the left suprarenal vein empties into the left renal vein, so that the left kidney is, to a certain extent, fixed to the immovable suprarenal capsule, while the right suprarenal vein empties into the inferior vena cava. The renal artery supplying the movable kidney is lengthened. The kidney again becomes fixed, in rare cases, by adhesions to adjacent organs.

2. Not infrequently this condition is unaccompanied by symptoms. Sometimes the patients detect a tumor in the abdomen. Or they complain of hysterical symptoms, pains in the abdomen, general malaise. The symptoms are often increased by vigorous bodily exercise, or in certain positions of the body, or they undergo exacerbations at the period of menstruation. Floating kidney may also give rise to obstinate jaundice (pressure on the ductus choledochus) or dilatation of the stomach (pressure on the duodenum).

The kidney is sometimes found within the superior strait of the pelvis, and may be moved to and fro over a considerable distance. The tumor is smooth, and the shape of the kidney can be recognized. In one

of my cases the renal artery could be felt pulsating in the hilus. The tumor is usually not very sensitive, though pressure in rare cases may give rise to nausea and vomiting.

A slight depression is sometimes noticeable over the region of the kidney, and this part is generally tympanitic on percussion. If the organ is replaced, the depression disappears and the tympanitic percussion note becomes dull. In some cases the tumor cannot be felt until the patient assumes an erect position or the knee-elbow position.

Disturbances of micturition may be entirely absent. In some cases there are occasional paroxysms of pain. The patients complain of intolerable pain, suffer from chills, fever, vomiting, sweating, and not infrequently go into collapse. The urine becomes scanty, and often contains pus or blood. Gilewski explains these symptoms by torsion of the kidney on its axis and occlusion of the ureter. Mosler showed that, in some cases, the symptoms are the result of acute inflammation of the kidney and its pelvis after occlusion of the ureter by plugs of mucus, fibrin, or blood, while in other cases they are the result of peritonitis. Orum repeatedly observed abortion as the result of floating kidney. Œdema of the feet is sometimes produced by pressure on the veins.

3. In many cases the condition cannot be differentiated from other abdominal tumors. Important factors in diagnosis are the changes in the region of the kidney, the shape of the tumor, and the recognition of pulsation at the hilus.

4. Treatment consists in the reposition of the kidney and fixation by means of a bandage. Iron and tonics are useful in some cases. If symptoms of incarceration appear, we may recommend enemata and narcotics. Nephrotomy is sometimes performed in this condition. Among fourteen cases collected by Billroth, six proved fatal. Hahn recommended nephrorrhaphy, *i. e.*, sewing the kidney to the abdominal walls. After a time, however, the newly-formed adhesions undergo relaxation and again permit the development of the disease.

APPENDIX.

Congenital abnormally low position of the kidneys (*dystopia renum*) is not infrequently observed. As a rule, this condition is associated with abnormal relation of the renal pelvis, ureter, and vessels. The left kidney is most frequently involved. It may be situated in the pelvis, and cause an obstruction to labor. In other regards the condition possesses no interest.

The dislocation is sometimes acquired, as in tumors of the liver and spleen, and in tight lacing. The right kidney is most frequently affected. Pressure of the dislocated kidney upon the duodenum and pylorus may give rise to vomiting and dilatation of the stomach. In some cases a floating kidney is retained in an abnormally low position by inflammatory adhesions.

14. *Anomalies in the Shape and Number of the Kidneys.*

a. The kidneys are sometimes found to be lobulated, being traversed by deep furrows. This is a partial retention of the foetal condition. It possesses no clinical interest, but must be differentiated from the lobulation resulting from syphilis and chronic interstitial nephritis. In the congenital form the capsule is unchanged; in the acquired form it is usually thickened and adherent over the furrows.

b. Horse-shoe kidney is the term applied to more or less complete union of the two organs. They unite most frequently at their lower ends, either by a fibrous bridge or coalescence of the renal tissue. The kidneys thus form a semicircular ring, with the convexity downwards.

In some cases the union takes place at the middle of the kidneys, most rarely

at the upper end. Occasionally the entire internal surfaces are adherent. The pelvis, ureters, and vessels are often abnormal.

This anomaly is sometimes recognized during life. A tumor is found in the abdomen, to which pulsations are sometimes conveyed by the aorta. If the kidney is situated very low, combined examination (abdomen, and vagina or rectum) should be made. The renal region proper is depressed, and furnishes a tympanitic percussion note. Symptoms are usually absent, but Langenbeck observed several cases in children who died of uræmia. Neufville observed compression and thrombosis of the inferior vena cava by a horse-shoe kidney. Cruveilhier reports abscess formation and perforation into the rectum.

c. Absence of one kidney has been described a number of times. The single kidney present is usually in a condition of compensatory hypertrophy. Three forms of this condition are recognized, viz.: absence of the kidney and ureter, absence of the kidney and upper part of the ureter, rudimentary kidney with developed ureter.

Absence of the kidney is sometimes acquired. Cases have been observed in which the organ was converted into a mass of fat. Evans described a case of almost complete disappearance of the left kidney from occlusion of the renal artery.

d. Supernumerary kidneys are less frequent than absence of a kidney; this condition has no clinical significance.

APPENDIX.

Aneurism of the renal artery is observed in rare cases. The following symptoms have been described: a pulsating tumor, renal pain, and hæmaturia. Finally, the aneurism ruptures and death ensues.

PART III.

DISEASES OF THE PELVIS OF THE KIDNEY AND THE URETERS.

1. *Hydronephrosis.*

I. ETIOLOGY.—The conditions necessary to the development of hydronephrosis are always furnished when the outflow of urine meets with an obstruction in the urinary passages. The urine then accumulates above the site of stenosis, gradually dilates the pelvis, and, by pressure on the papillæ, entirely prevents the escape of urine from the kidney. But if the mucous membrane of the renal pelvis continues to produce secretion, its distention may increase, and the kidney gradually may undergo atrophy from the increasing pressure.

Hydronephrosis may be congenital or acquired. The former may constitute an obstruction to delivery, or may interfere with the movements of the diaphragm. We will discuss only the acquired form of the disease.

It may be unilateral or bilateral, the former variety being more frequent. The right side is affected oftener than the left.

In total hydronephrosis the entire pelvis is dilated, in partial hydronephrosis only a portion of the pelvis.

Partial hydronephrosis is rare. Heller reported a case of double pelvis, in which only one pelvis was dilated. Fenge observed an abnormal valve in the middle of the renal pelvis, with obstruction to the escape of urine from one part of the pelvis.

Finally, hydronephrosis may be temporary (intermittent) or stationary. In the former, the symptoms increase at times, as the obstruction becomes greater.

The causes of stasis may be situated in any part of the urinary passages. They include: renal calculi with occlusion of the ureter or injury of the mucous membrane with subsequent cicatrization, compression of the ureter by tumors of the kidney or its pelvis, or echinococci, floating kidney with sudden flexion of the ureter, abnormal origin of the renal artery with pressure on the ureter, peritonitic and paranephritic exudations or connective-tissue proliferations with pressure on the ureter, vesical calculi and tumors, urethral stricture, phimosis, hypertrophy of the prostate, tumors of the ovary and uterus, etc.

In some cases the stasis is the result of the abnormal entrance of the ureter into the renal pelvis, and the unusual development of valves at the entrance or outlet of the ureter. Such conditions can only be recognized if the organs are left in situ. It must also be remembered that abnormal valves are often constructed in such a manner that they permit the passage from the ureter to the renal pelvis, but not in the opposite direction.

The disease occurs especially in middle life, and is more frequent in females.

II. ANATOMICAL CHANGES.—In well developed cases the kidney and its pelvis are converted into a large sac filled with fluid; it may exceed the dimensions of a man's head. The surface of the sac is usually nodular and lobulated. The individual lobulations correspond to the dilated calices. In other cases we notice scattered remains of the renal parenchyma. Not infrequently, the kidney forms a flattened mass in which the renal structure (with marked interstitial proliferation of connective tissue) is distinctly visible.

The gradual disappearance of the kidney takes place in the following manner: the papillæ are first flattened by the pressure, then the medullary substance disappears, finally the cortex is involved. In some cases the kidney does not atrophy from simple pressure, but as the result of ulcerative processes.

The hydronephrosis is generally very adherent to adjacent loops of intestines; the adhesions are sometimes as firm as cartilage. If the obstruction is situated in the lower part of the ureter, the latter is dilated, sometimes to the size of the small intestine.

The inner surface of the sac is usually smooth, and is sometimes lined with a deposit of urates or phosphates.

The amount of fluid may exceed thirty litres. It is light-yellow, thin, and clear, or reddish-brown (extravasations of blood); in rare cases it has a colloid or gelatinous consistence. Caseation and calcification of the contents are rarely observed. Glistening crystals of cholestearin are found occasionally. Under the microscope we find epithelium, granular cells, red blood-globules, round cells, occasionally shreds of renal tissue.

The chemical constitution depends upon the age of the hydronephrosis. In old cysts the urea and uric acid disappear entirely. Bérard gives the following analysis in one case (per 1,000 parts).

Water.....	920
Albumin....	75
Salts and extractive matters ..	4
Organic substance	1
Urea	0

In one case Woelfler found urea, uric acid, creatinin, and indican. In Schetelig's case the fluid was colloid, and contained no urea, but paralbumin, mucin, serumalbumin, and cholestearin, so that a diagnosis of ovarian tumor was made.

III. SYMPTOMS.—The main symptom is the demonstration of a fluctuating renal tumor, and the exclusion of abscess, echinococcus, or tuberculosis of the kidney. The dilated ureter can sometimes be felt through the abdominal walls. In unilateral hydronephrosis no changes are produced in the constitution of the urine.

The patients often complain of a feeling of tension, dyspnœa, and constipation—the results of compression of the diaphragm and intestines. These symptoms do not develop until the tumor has attained considerable size. The colon is often situated over the tumor.

Slight distention of the renal pelvis remains entirely latent, or is discovered accidentally during careful examination of the abdomen. It is sometimes found only in certain positions of the body, especially the upright position. Our attention is sometimes attracted by sudden chill, fever, vomiting, and violent pain—the results of rapid occlusion of the ureter by calculi, etc. In such cases, increase and diminution of the tumor have been observed as signs of temporary hydronephrosis.

Hæmaturia has been observed in these cases after injury to the abdomen. Rupture of the sac may also take place, and be followed by diffuse peritonitis. In one case, Dittel observed spontaneous rupture of the sac and corresponding renal artery. Injury may also be followed by suppuration and its symptoms (chills, sweats, diarrhœa, etc.). Perforation sometimes takes place into the intestines, peritoneal cavity, pleura, lungs, bronchi, or through the muscles of the loins.

IV. DIAGNOSIS.—In the first place, we must recognize the presence of a tumor of the kidney (vide page 303). The diagnosis of hydronephrosis would be favored by the demonstration of an obstruction to the escape of urine. In renal echinococcus we must search for hydatids in the urine, in tuberculosis for tubercle bacilli in the urine. In renal cancer the signs of cachexia are noticeable; in renal abscess attention must be directed to the development and course of the disease. An exploratory puncture may be made, but it should be remembered that this not infrequently gives rise to peritonitis. Hydronephrosis has often been mistaken for ovarian tumors, and therefore a careful examination per rectum should be made in all cases.

V. PROGNOSIS.—This is not unfavorable when the primary disease can be relieved. The results of operation heretofore have not been good.

VI. TREATMENT.—Whenever possible, treatment should be directed against the primary disease (phimosis, stricture, renal calculi, etc.). In some cases, after removal of the obstruction, it is advisable to make careful pressure on the pelvis of the kidney, in order to drive the urine into the bladder.

If the causal indication cannot be met, the cyst may be removed by operation. This often meets with unconquerable obstacles on account of the numerous adhesions to adjacent loops of intestines.

If symptoms of incarceration appear, we may order warm poultices to the renal region, make injections of morphine, and, if necessary, replace displaced organs.

2. *Pyelitis.*

I. ETIOLOGY.—Inflammation of the pelvis of the kidney may be primary or secondary, acute or chronic, catarrhal, purulent, or hemorrhagic.

Hemorrhagic pyelitis occurs in acute exanthemata which have assumed a hemorrhagic character, and in conditions of blood dissolution. The mucous membrane of the renal pelvis is injected and covered with more or less numerous extravasations of blood. Extensive bloody suffusions are sometimes found in the submucous tissue. In some cases, the pelvis contains coagulated blood. Under the term *pyelo-néphrite hémato-fibrineuse*, Ollivier has described a hemorrhagic form of pyelitis which is peculiar to old age, and is associated with arterio-sclerosis, the formation of aneurisms in the branches of the renal artery, and hemorrhages from the latter. Hemorrhagic pyelitis possesses no clinical significance.

Catarrhal and purulent pyelitis, which will alone be considered, are rarely primary. Cold and injury have been mentioned as causes; in many cases no cause can be discovered.

As a rule, pyelitis is secondary. It is generally the result of mechanical irritation by foreign bodies, particularly calculi, much more rarely, echinococci, cast-off shreds of renal cancer or tubercles, parasites in the renal pelvis (*strongylus gigas*), or blood clots.

Next in frequency among the etiological factors are conditions of urinary stasis. Simple stasis may produce pyelitis in a purely mechanical manner, but more intense inflammation is produced if the urine undergoes ammoniacal decomposition. Among the causes of urinary stasis may be mentioned: phimosis, urethral stricture, cystitis, paralysis of the bladder, etc.

Stasis catarrh occurs very often in females during pregnancy and parturition, and after gynæcological operations. Kaltenbach and Stadtfelt explain this fact by the pressure of the uterus or enlarged pelvic organs on the ureters.

In some cases the inflammation extends from adjacent parts, as in paranephritis or inflammatory processes in the kidneys. Gonorrhœa may also give rise to pyelitis.

Pyelitis is sometimes the result of toxic agents (copaiba, cubebs, turpentine, etc.). Finally, it occurs during infectious diseases (typhoid fever, cholera, diphtheria, small-pox, etc.). Ritter and Huettenbrenner observed it frequently in acute enteritis of infancy. The causes of pyelitis in diabetes mellitus are unknown.

The disease is more frequent in middle life. It is more common in men than in women.

II. ANATOMICAL CHANGES.—In acute pyelitis the mucous membrane of the pelvis of the kidney is very congested, loosened, and less glossy. The redness is uniformly diffuse, or appears as individual dilated vessels; extravasations of blood are observed not infrequently. The loosening depends mainly on serous infiltration of the submucous layer, while the diminished gloss is the result of desquamation of the epithelium cells. The surface is often covered with a gelatinous, mucoid fluid, which sometimes has a purulent appearance from the profuse admixture of pus-corpuscles.

Losses of substance are seen occasionally in the mucous membrane. These may lead to perforation into the paranephritic tissue, peritoneum, pleura, lungs or bronchi.

In chronic pyelitis the mucous membrane has a brownish-red color, occasionally a slate color. We sometimes find a number of almost poly-poid proliferations, or the mucous membrane is coated with a gritty layer of urates and ammonia-magnesia phosphates. Ulcerations may also be present. The wall of the pelvis is often considerably thickened.

These changes are very commonly associated with dilatation of the renal pelvis, either as a result of the primary disease, or because urinary stasis is produced by plugs of mucus and pus, and by swelling of the inflamed mucous membrane of the ureter. The hydronephrosis may form an extensive tumor, the pressure of which causes atrophy of the substance of the kidney.

Pyelitis is associated not infrequently with purulent nephritis (pyelonephritis). This is observed most frequently in the pyelitis which follows stasis and ammoniacal decomposition of the urine (vide p. 290). In such cases there may be extensive losses of substance in the renal parenchyma, so that the kidney and its pelvis sometimes form merely a sac filled with pus (pyonephrosis).

The purulent masses in the renal pelvis sometimes become thickened, and undergo caseation and calcification. This results finally in atrophy of the kidneys, obliteration of the pelvis and ureter; the unaffected kidney undergoes compensatory hypertrophy.

FIG. 90.



Epithelium cells of the mucous membrane of the renal pelvis, with prolongations and a shingled arrangement, from the intact mucous membrane in man. Preparation macerated in chromic acid. Enlarged 275 times.

III. SYMPTOMS.—Many cases of pyelitis are unrecognized during life on account of the prominence of the symptoms of the primary disease. Pure cases will present the following clinical history:

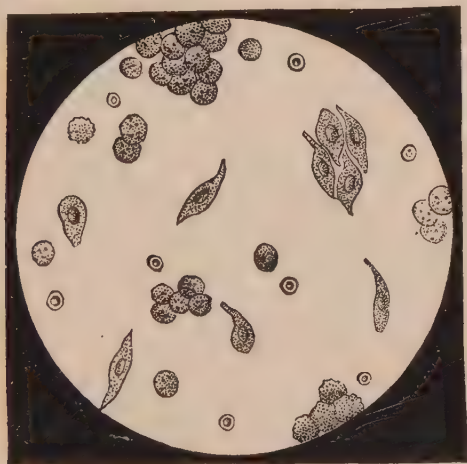
The patients complain of pain in the region of the affected kidney. It is sometimes a dull feeling of pressure, sometimes a very violent pain, which radiates along the ureter toward the mons veneris, perineum, and thigh. In our experience the pain is the result of tension, and is so much more violent the more rapidly the symptoms develop and the more marked the accompanying hydronephrosis. In calculous pyelitis, the mechanical irritation of sharp calculi must also be taken into consideration.

The pains are accompanied by vesical tenesmus. The patients experience the desire to empty the bladder at short intervals, but often pass only a few drops at a time.

The urine is acid, the color and specific gravity are unchanged; it contains pus and an increased amount of mucus. The albumin contained in the urine corresponds to the amount of pus. Oppolzer lays stress on the fact that, as a rule, the quantity of urine is increased, and suggests that some cases of diabetes insipidus are merely unrecognized inflammations of the renal pelvis.

If a large amount of pus is present, the sediment may form one-quarter of the entire volume of the urine. It consists mainly of pus-corpuscles. Special importance has been attached to the presence of epithelium cells from the pelvis; in the deeper layers these present prolongations and a shingle-like arrangement (vide Figs. 90 and 91). But, in the first place, these cells are not found very often in the sediment, and, in addition, they are almost identical in appearance with the deeper epithelial layers of the bladder. The sediment always contains a few red blood-globules.

FIG. 91.



Urinary sediment in acute pyelitis, containing epithelium, round cells, and red blood-globules. Enlarged 275 times.

If pyelitis is associated with hydronephrosis, a renal tumor may become noticeable. Its increase or diminution in size is observed not infrequently according as the urinary passages are more or less obstructed. Enlargement of the tumor is sometimes associated with severe pain, chill, fever, and vomiting, and the urine becomes clear and free from pus. As these symptoms subside, diuresis becomes more profuse, and the urine grows cloudy. This is evidently the result of temporary complete occlusion of the outlet from the pelvis of the kidney by pus, mucus, calculi, parasites, shreds of a tumor, or clots of blood. Under such circumstances, Ebstein found fat in the urine and hæmatoidin in the sediment.

The history of pyelitis may be confined to the symptoms mentioned above, though, as a rule, other symptoms are noticed.

If the disease is complicated by nephritis, the amount of albumin in

the urine increases, and casts are also found in the sediment. In some cases we find the symptoms of suppurative nephritis (vide p. 292).

If the pyelitis is the effect of calculi, hemorrhages and attacks of renal colic occur frequently, and the sediment of the urine contains various crystalline constituents.

When the disease is caused by parasites, attention should be directed to their evacuation in the urine; moreover, sudden symptoms of occlusion of the ureter are not infrequent in such cases.

Finally, if pyelitis is the result of stasis and decomposition of urine, the symptoms may be altogether masked by those of chronic cystitis.

The duration of the disease depends upon the individual causes. If the latter are temporary in their nature, the pyelitis will be acute, *i. e.*, it may last two to six weeks; in other cases it continues months and even years.

The disease may terminate in complete recovery, or the amount of pus produced is so great that the patients become weak and cachectic, dropsy develops, and they finally die in collapse. Death is sometimes the result of anuria and uræmia. This occurs when the ureters are occluded by calculi. Indeed, the occlusion of one ureter sometimes suffices to render the unaffected kidney incapable of function (reflex irritation). In ammoniacal decomposition of the urine, death may occur from ammoniæmia. Finally, a fistula may be produced, and may open into the loins, intestines, peritoneum, pleura, or air passages.

IV. DIAGNOSIS.—The diagnosis of pyelitis is often extremely difficult. If there is pus in the urine, we must decide whether it comes from the kidneys, from perforating paranephritic abscesses, the bladder, prostate gland, urethra, or pelvis of the kidney. In diseases of the kidney and paranephritic abscesses a tumor will be found in the renal region; in vesical affections the reaction of the urine is alkaline; in inflammation of the prostate the gland is swollen, reddened and painful; if the pus is derived from the urethra it is generally small in quantity, and usually makes its appearance after pressure upon the urethra.

It is especially difficult to make a diagnosis of pyelitis, if it is associated with disease of the kidneys or bladder.

V. PROGNOSIS.—This depends upon the curability of the primary disease.

VI. TREATMENT.—This embraces prophylactic, causal, and symptomatic measures.

Prophylactic measures include caution in the use of balsams and powerful diuretics, and the most scrupulous cleanliness of the instruments employed in catheterization.

Causal treatment includes the measures adopted in phimosis, urethral stricture, cystitis, renal calculi, etc.

In acute pyelitis the patient should be kept in bed. The diet should be mainly fluid and unirritating (lukewarm milk mixed with lime-water, weak tea, bouillon, eggs, tender meat, diluted claret). If the patient suffers from great thirst, we may order carbonated waters.

A warm poultice should be applied constantly over the affected kidney, and subcutaneous injections of morphine made in the region of the kidneys if the pains are violent.

In many cases this plan of treatment will suffice. If the production of mucus and pus is very abundant, we may employ astringents:

℞ Acid. tannic.....gr. ivss.
 Opii puri.....gr. ʒ
 Sacch. alb.....gr. viij.

M. Sig. One powder to be taken every three hours.

In addition, or alternating with the above prescription, we may give:

℞ Fol. uvæ ursi..... ʒ iiij.

D. S. To be taken daily as a tea in four cups of water.

Many physicians extol the use of the balsams (turpentine, copaiba, tolu, cubebs, etc). Maskaj obtained good results from tincture of cantharides (twenty-five drops, t. i. d.).

The treatment of chronic pyelitis is almost identical with that of the acute form. We may particularly recommend the alkaline waters (Vichy, Bilin, Geissshuebel, etc.), or the alkaline-muriatic acidulous wells (Ems, Selters, Salzbrunn).

If the kidney and its pelvis are entirely destroyed by suppuration, they may be removed by operation. Among forty operations collected by Billroth, eighteen proved fatal.

3. Renal Calculi. Nephrolithiasis.

I. ETIOLOGY.—According to the situation in which renal calculi are found they are known as calculi of the kidney, renal pelvis, ureter, bladder, or urethra. They have even been found in the dilated prostatic sinus. It is very common that stones which have formed in the pelvis of the kidney pass into the bladder, and there become vesical calculi.

The calculi are formed most frequently in the renal pelvis, rarely within the kidney, and only in exceptional cases in the ureters. Next in frequency to the pelvis of the kidney as the site of the formation of calculi stands the bladder.

In the following remarks we will discuss only that variety which forms in the pelvis of the kidney. They sometimes extend deep into the substance of the kidney, and in Maring's case even reached the surface of the organ.

Renal calculi are most frequent in childhood and old age. They have been found even in the foetus. They are most frequent from the ages of two to twelve years, but are not as common at this period as stone in the bladder.

The calculi are more frequent in the male than in the female sex (in the proportion of three to one).

Sedentary habits, especially if associated with high living, favor their development. They are more frequent, therefore, among the well-to-do.

Telluric conditions also exert a certain influence. Thus the disease is unusually frequent in England and Holland. Striking contrasts are sometimes observed in localities which are situated very close together. No satisfactory explanation of this feature has yet been offered.

A rare case has come under my observation in which a distinguished surgeon is able, with absolute certainty, to produce renal calculi in himself by drinking white wine. Even a quarter of a wineglassful is sufficient. At the end of a few hours violent pains are experienced, and finally calculi are voided. They may attain the size of a pea, and are composed exclusively of uric acid; symptoms of calculi are never produced if the ingestion of white wine is avoided.

Heredity plays a part in certain cases, and occasionally the stones

even possess the same chemical constitution. This is particularly true with regard to cystin calculi. It is evidently the result of the inheritance of abnormal nutritive processes.

Certain individuals seem to possess a predisposition to the formation of calculi, and in such cases earthy deposits are also found in the bile-ducts, salivary ducts, etc.

In rare cases, injury to the renal region has been regarded as the immediate cause.

Diseases of the kidney and its pelvis, if associated with retention and decomposition of the urine, may undoubtedly give rise to the formation of calculi.

Certain diseases of nutrition favor their development. For example, uric-acid calculi are not uncommon in gout, and phosphatic stones have been found in a number of cases of osteomalacia.

II. ANATOMICAL CHANGES.—As a rule, calculi are found in the pelvis of one kidney alone.

According to the size of the concretions we distinguish renal gravel and calculi. Gravel is a powdery, finely granular deposit, the particles not exceeding the size of a pin-head. It is almost always composed of uric acid and its salts.

The number of renal calculi varies between very large limits. Gee recently observed a case in which the dilated right pelvis contained, in addition to a considerable amount of gravel, nearly a thousand calculi. The other pelvis was also filled with numerous stones, one of which was unusually large and heavy.

Calculi have been described which were larger than a goose egg. Of course, in such cases the pelvis is dilated, and sometimes the parenchyma of the kidney undergoes atrophy. In Gee's case, referred to above, the large calculus weighed thirty-six ounces. The specific gravity varies from 1.200 to 1.930.

The stones may be round, elongated, angular, or entirely irregular. In some cases the calculi are found, not in the renal pelvis, but in one or more calices (always in the deepest ones). If the calculus fills the entire pelvis and all the calices, it has an irregularly branched shape, like that of a deer's antlers.

There are seven varieties of renal calculi: *a.* those composed of uric acid and urates; *b.* those composed of oxalate of lime; *c.* those composed of phosphates; *d.* those composed of carbonate of lime; *e.* those composed of cystin; *f.* those composed of xanthin; *g.* those composed of indigo.

In certain cases we also find concretions of fibrin, the remains of previous hemorrhages into the renal pelvis. They usually have a leathery consistence, and burn with a yellow flame which smells like burnt feathers.

This classification cannot be adhered to strictly, since the calculi are sometimes composed of more than one chemical substance. Upon sawing through the stone, we find a nucleus (rarely several), around which are deposited peripheral layers. The nucleus is generally composed of uric acid, the peripheral layers of oxalate of lime, phosphates, or alternately of this or that substance. In some cases the stone consists of a nucleus, body, and a thin peripheral cortex.

Ebstein showed that the calculi are held together by an organic framework composed of an albuminoid substance.

The most frequent forms of calculus are those composed of uric acid and urates. They are only exceeded in hardness by oxalate of lime calculi. Their surface is sometimes smooth and almost as if polished; sometimes it is dotted with fine nodules and granules. The surface of fracture is generally granular and amorphous, more rarely it is crystalline or leafy. As a rule, the stone is laminated, lighter and darker rays alternating; the individual layers sometimes run a wavy course. Their color varies from light-yellow to brown or brownish-red. The nucleus consists, in not a few cases, of oxalate of lime, or the nucleus of uric acid is surrounded by layers of oxalate of lime. Lamina of uric acid and oxalate of lime sometimes alternate with one another; the surface is not infrequently covered with a thin layer of earthy phosphates.

Renal calculi, which consist almost exclusively of acid urate of ammonia (with a trace of oxalate of lime), are found in infants. These stones are usually small, light-brown, brittle, and lose their color when dry.

The presence of uric acid in calculi can be determined by means of the murexide test. A little powder is scraped from the stone into a porcelain dish, a drop of nitric acid is added, and then heated until dry. Light-brown spots will then form. If these are touched with a drop of ammonia, a beautiful carmine-red, so-called murexide color (acid purpurate of ammonia), will form if uric acid is present. If a drop of liquor potassæ is now added, the color changes to a dark violet-blue. In contradistinction to xanthin compounds, the color grows pale on being warm, and disappears entirely before it is dry.

In order to detect urate of ammonia, the stone should be pulverized and then covered with hot water. The latter will dissolve considerable urate of ammonia, but very little uric acid. When the water grows cold, the urate of ammonia is precipitated. If the latter is now boiled with potash, the odor of ammonia is given off, turmeric paper, which is held in the vapor, is colored brown, and a glass rod, which is moistened with acetic acid and held over the vapor, gives off a white cloud of acetate of ammonia. If this reaction remains absent while the murexide test gave negative results, the calculus is composed of uric acid alone.

Calculi of oxalate of lime are very hard, and have a very rough, warty surface (mulberry calculi). In rare cases they are colorless; usually they have a very dark, brown, gray, or blackish-brown color. Their surface of fracture is amorphous; it rarely contains small octahedra or dumb-bell crystals.

Pure oxalate of lime calculi are not common. The nucleus sometimes contains uric acid, or layers of uric acid and oxalate of lime alternate with one another. The peripheral layers are composed not infrequently of earthy phosphates. Oxalate of lime calculi are sometimes found in the pelvis of one kidney, uric-acid calculi in the other.

Oxalate of lime calculi become white when heated to a glow. Carbonate of lime is first formed (this effervesces on the addition of acids), under more intense heat caustic lime is formed, which turns moistened turmeric paper brown. The stone is unaffected by acetic acid, but dissolves without effervescence in mineral acids.

Phosphatic calculi are composed of phosphate of lime and ammonia-magnesia phosphates (triple phosphates).

Calculi which are composed solely of the triple phosphates occur very rarely. They are small, with a rough surface, easily compressed, whitish in color, and have a crystalline surface of fracture. Triple

phosphates sometimes form the cortex, more rarely the nucleus of gall-stones.

As a rule, phosphatic calculi form a combination of phosphate of lime and triple phosphates. The less the proportion of the latter the less brittle are the stones. Their surface is rough, the surface of fracture granular, their color gray, clay to violet-red.

When heated to a glow these calculi form a white, enamel-like mass. If the powder is then dissolved in hydrochloric acid, and ammonia is added, a precipitate is formed. Triple phosphate calculi emit, when heated, an odor of ammonia.

Carbonate of lime calculi are very common in man. They form small stones of a whitish, brownish, or violet color, with an earthy surface of fracture. Carbonate of lime is sometimes found in phosphatic calculi.

Lime calculi effervesce on the addition of hydrochloric acid.

Cystin calculi are rare. They are sometimes formed entirely of cystin, sometimes the nucleus is composed of uric acid, or they may be mixed with small amounts of the earthy phosphates. Their surface is sometimes smooth, sometimes rough. The surface of fracture has a lamellar structure. Their color is dull white or amber-yellow, more rarely greenish-gray; they sometimes assume an emerald green or even ultramarine blue color after exposure to the air.

Cystin dissolves in potash or ammonia, and, after evaporation, deposits the well known six-sided crystals of cystin (vide Fig. 70).

Xanthin calculi are found very rarely. They have a smooth, shining surface, laminated structure, and a yellowish-brown to dark-brown color. The surface of fracture on being rubbed has a waxy gloss.

To detect xanthin, the powdered stone is placed in a porcelain dish, a drop of nitric acid is added and then heated to dryness. The powder then assumes a citron-yellow color. The color remains unchanged if ammonia is added, but turns red on the addition of potash.

An indigo calculus was found by Ord in a kidney which was partly destroyed by sarcoma. The stone weighed nearly an ounce and a half, had a bluish-black surface, and made a bluish-black mark on paper. In addition, it contained phosphate of lime and blood clots.

On being heated, the powder gave off purple-red vapors, which precipitated in the shape of dark blue microscopic prisms. On the addition of concentrated sulphuric acid a dark brown solution formed, which turned opaque blue in a few days. After being diluted with water, this gave the yellow line peculiar to indigo in the spectrum.

If there is a communication between the biliary and urinary passages, the latter may be found to contain gall-stones.

Renal calculi almost always give rise to calculous pyelitis. This may be catarrhal or purulent; in rare cases it is attended with necrotic changes. Ulcerations sometimes form, and may perforate into the stomach, intestines, pleura, bronchi, or externally through the loins. Perforation into the peritoneum may also take place, and rapidly prove fatal.

The pyelitis often extends to the parenchyma of the kidney, and gives rise to interstitial changes or the formation of abscess in the kidneys. Finally, the kidney and its pelvis are converted into a multilocular sac filled with stinking pus and calculi. In some cases the renal tissue has disappeared almost entirely. A combination of nephrolithiasis and Bright's disease is observed not infrequently.

If the process is unilateral, the other kidney undergoes compensatory hypertrophy; after long protracted suppuration, it undergoes waxy changes.

When renal calculi begin to migrate, there is danger that they will remain in the ureter. The urine then collects above the occluded spot (acute hydronephrosis). Occasionally there is an outlet at the side of the calculus for the escape of urine, or sometimes the calculus is situated in a sort of lateral groove which permits the passage of urine to the bladder. If the stone is unable to move upwards or downwards, ulceration of the ureter is apt to develop, and may terminate in perforation. The ureter may also undergo rupture.

The calculi sometimes attain such large dimensions that a renal tumor may be recognized during life.

Catarrhal cystitis, urethral stricture, hypertrophy of the prostate, phimosis, etc., are often associated with renal calculi.

III. SYMPTOMS.—In not a few cases, no symptoms are observed during life. In other cases the calculi are passed unexpectedly in the urine, although no previous symptoms have been noticed.

Some patients complain of gastric symptoms. They suffer at times from attacks of nausea, vomiting, and pain in the stomach. In other cases the calculi are marked by the symptoms of cystitis. Much more frequently we find the symptoms of pyelitis, whose etiology is cleared up after the passage of gravel or calculi, or the detection of crystalline deposits in the urinary sediment. The latter are important, inasmuch as they enable us to judge the nature of the calculus, but it must be remembered that calculous pyelitis may last a long time, although the urine never contains a crystalline sediment.

Repeated hæmaturia is very often the sole symptom of renal calculi. This symptom, if associated with violent pains, points toward renal calculi. The hemorrhage is sometimes very profuse, and may consist of fresh blood. Cylindrical clots of blood (casts of the ureter) are sometimes passed.

In extremely rare cases, the stones are so large as to form a palpable renal tumor, and to give rise to a grating sensation when they are rubbed against one another.

Renal colic is one of the most important symptoms of the disease, but it occurs only when the calculi meet with obstruction in their passage through the ureter. The pains sometimes occur unexpectedly during sleep, sometimes they follow bodily exertion or mental excitement. They may also follow a fall or blow in the region of the kidney, tight lacing, or the act of coitus.

The most prominent symptom is pain. It is often excruciating, so that the patient sinks into a condition of collapse. It is generally localized, first in the region of the kidney, then about the middle of the ureter, later in the bladder. It usually radiates towards the shoulder, testicle, thighs, even into the epigastric region. The cremaster muscle is often contracted spasmodically, and the testicle drawn upwards; in rare cases the testicle is swollen. There is often a feeling of formication, coldness,

and weakness in the affected leg. The pains are sometimes so violent as to give rise to syncope, or, in children, to general convulsions. The pains are generally unilateral. In some cases, they cease abruptly, either because the stone has passed backwards into the pelvis of the kidney, or because it has entered the bladder. In the former event, the pains reappear when the stone again enters the ureter; in the latter event, they will recur if the passage through the urethra meets with obstacles. In certain cases the pain ceases only for a short time, while the calculus is passing freely through a certain portion of the ureter, and then returns as soon as it again becomes impacted. There is, as a rule, unusual difficulty in passing through the opening of the ureter into the bladder.

The onset of the pain is often attended with a chill, fever, and vomiting. The patients usually lie upon the affected side, the spine and thighs being strongly flexed. Even irrespective of the attacks of colic, the patients not infrequently hold the spine peculiarly stiff and bent over forwards.

The sufferers complain of vesical tenesmus, and are often able to pass only a few drops of urine at a time. The urine often contains blood, because the sharp calculi injure the mucous membrane. If pyuria has been present, the urine frequently becomes clear during the attack of colic, because the pus-containing urine cannot enter the bladder on account of the occlusion of the ureter. Total anuria is sometimes observed; for example, when both ureters are occluded by calculi, or if there is only one kidney. But even unilateral occlusion may abolish, by reflex action, the function of the kidney on the opposite side. Anuria is sometimes the effect of occlusion of the urethra. As a matter of course, anuria puts the patient in great danger of death from uræmia. Paget reports a case in which the first uræmic symptoms did not appear until the anuria had lasted two weeks; in other cases, they develop at the end of the first day. But all hope should not be abandoned, even under such circumstances. Russel observed recovery in a case in which anuria had lasted twenty-eight days. If the obstruction to the escape of urine is suddenly removed, very large quantities of urine are sometimes passed in a very short time.

The region of the kidneys is often very sensitive to pressure, and occasionally we can detect an acute dilatation of the renal pelvis and the ureter. Calculi which have been impacted in the ureter can sometimes be palpated from the vagina or rectum, or by means of the catheter.

In pregnant women, renal colic often produces abortion. Troja reports a case in which a woman aborted fourteen times in consequence of renal colic.

Calculi are not infrequently found in the urine after the attack; but, as they may remain in the bladder for a considerable period, the examination of the urine should be continued for several days.

If the stone remains impacted in the ureter, or ulcerative processes occur in the renal pelvis, perforation into the peritoneal cavity may take place, and will be followed by the signs of a rapidly fatal, acute peritonitis, or a communication with the stomach, intestines, or air passages may be formed, or a paranephritic abscess may develop and rupture externally.

Attacks of renal colic usually recur, and the disease often lasts for years.

Renal gravel gives rise to much less serious symptoms. The pains

in the region of the kidney are not as severe as in renal colic. The urine contains a crystalline sediment, but sometimes the sediment accumulates in the urethra and obstructs the escape of urine.

The causes of the formation of gravel and calculi are, in great part, unknown. We may mention the following possibilities: (*a*) Constituents of the normal urine are formed in such quantities that they no longer remain dissolved, but are precipitated; (*b*) Abnormal substances, such as cystin, pass into the urine and form concretions; (*c*) The urine undergoes decomposition and deposits various substances, according as the decomposition is acid (doubted by the majority of recent writers) or alkaline; (*d*) Precipitates form around foreign bodies; thus, in Egypt, Griesinger found the ova of hæmatobia as nuclei of renal calculi; (*e*) Meckel suggested that a specific catarrh of the mucous membrane (calculus-forming catarrh) was the cause of the formation of calculi.

It is evident that various factors must be taken into consideration in the development of calculi. The investigations of Ebstein show that epithelial catarrhs, which are hardly recognizable clinically, play an important part in the production of renal calculi, inasmuch as they furnish their organic binding substance.

Ultzmann differentiates primary and secondary calculi-producers. The former include uric acid, urate of soda, oxalate of lime, and cystin, *i. e.*, substances which are precipitated in acid urine. The latter include those substances which are deposited after alkaline decomposition of the urine, viz., urate of ammonia, phosphate of lime, and triple phosphates. Calculi are composed most frequently of primary stone-producers. But if these have given rise to suppuration and alkaline decomposition of the urine, they may gradually be converted into calculi which are composed of secondary stone-producers.

IV. DIAGNOSIS.—When the symptoms of pyelitis are present, the diagnosis of calculus-pyelitis is probable, if the sediment contains numerous crystals which have been passed in the urine. Ultzmann claims that the spear-shaped crystals of uric acid favor the diagnosis of renal calculi (*vide* Fig. 92).

In order to diagnose renal calculi from the presence of hæmaturia, we must be able to exclude injury, acute nephritis, passive congestion, embolism, cancer, echinococcus, and tuberculosis of the kidneys.

Renal colic may be mistaken for:

- (*a*) *Lumbago*: urinary changes are absent.
- (*b*) *Cancer or tuberculosis of the spine*: the pain is confined chiefly to the spinal column.
- (*c*) *Paranephritic abscess*: there is diffuse infiltration of the renal region.
- (*d*) *Embolism of the renal artery*: the source of embolism (usually valvular disease of the left heart) can be demonstrated.
- (*e*) *Biliary colic*: jaundice is generally present, and the pain is confined chiefly to the right anterior surface of the abdomen.

The nature of the calculus can usually be determined from the urinary sediment.

V. PROGNOSIS.—Although renal calculi rarely give rise to serious danger, the prognosis with regard to their removal is not very favorable, and a relapse may be looked for at any time.

VI. TREATMENT.—Among prophylactic measures the regulation of the diet is the most important. Those individuals who eat too much meat, exercise very little, and drink large amounts of strong wine and beer

should eat largely of vegetable food, exercise vigorously, drink weak tea and carbonated waters. An excessive vegetable diet may also prove injurious, since the salts of the vegetable acids are converted into carbonates within the body, the urine becomes alkaline, and may thus give rise to the deposit of secondary stone-producers within the urinary passages. These rules are particularly important in families in which obesity and gout are hereditary.

It is doubtful, at the least, whether calculi can be dissolved within the urinary passages, but the methods of treatment adopted to secure this end are the same as those employed to prevent their formation.

The previously-mentioned dietetic measures hold good in the treatment of uric-acid calculi. In addition to alkalies (sodium bicarbonate

FIG. 92.



Spear-shaped crystals of uric acid in calculous pyelitis. After Ultzmann.

and carbonate, potassium carbonate, sodium phosphate), fruit and grape cures have also been recommended as solvents. Benzoic acid and lithium enjoy special repute as solvents of uric acid. It must be remembered in practice, however, that all preparations of lithium are, as a rule, poorly tolerated by the stomach.

Mineral water cures enjoy a great repute in this disease, although many waters merely act as does the ingestion of large amounts of warm water, associated with proper diet and bodily exercise. Alkaline, acidulous-alkaline, saline wells, and lithium-containing waters have been recommended. The alkaline-acidulous wells include Bilin, Ems, Fashingen, Vichy, Salzbrunn, etc., the alkaline saline wells include Carlsbad, Marienbad, and Tarasp; the lithium wells include Assmannshausen, Baden-Baden, Duerkheim, etc. The patient should drink the water at

the wells for one or two months, and then continue the treatment at home for months, with occasional intervals of a few days or weeks.

In oxalate calculi the dietetic rules remain the same; those articles should especially be avoided which are rich in oxalic acid, for example, black tea, cocoa, sorrel, spinach, rhubarb. Among mineral waters we may particularly recommend simple acidulous wells (Selters, Apollinaris, etc.), or alkaline acidulous wells. The same treatment may be adopted for cystin calculi.

In phosphatic calculi, mineral or vegetable acids have been recommended, for example, hydrochloric, phosphoric, and lactic acids, and carbonated mineral waters.

If renal colic sets in, an injection of morphine may be made in the region of the kidney, or chloral hydrate, 3 ss.-i., given in a wineglassful of sugar-water. A warm poultice should be applied to the region of the kidney, and, if necessary, a warm bath lasting one-half hour to one hour, may be taken. The passage of the calculus is sometimes accelerated by stroking with the fingers along the course of the ureter. If the pains continue, the patient may be anæsthetized with chloroform. Simpson recommended that the patient stand on his head, in order to force the calculus back into the pelvis of the kidney. The bowels should be kept open.

Operative removal of the calculi has been performed successfully in a number of cases. The indications consisted of the formation of external fistulæ, or extremely violent suffering. As a matter of course, we must make a positive diagnosis in such cases, and satisfy ourselves that the other kidney is intact.

APPENDIX.

RENAL INFARCTIONS.—Crystalline deposits—so-called renal infarctions—are sometimes found in the tubules of the kidney. They possess slight clinical interest.

a. Uric-acid infarctions are found in infants who die during the first or second week of life. They are sometimes seen as late as the sixth week, or even the third month. As a rule, they are observed only in those children who have breathed for several hours after birth, so that they possess a certain forensic importance. They are found in about fifty per cent of the bodies, and have also been observed in a few cases in which the children had not breathed.

Upon section of the kidney we notice, near the papillæ, yellow, orange-yellow, or brick-red streaks, which radiate from the apices of the papillæ. In rare cases they extend through the entire medullary substance. On pressure, the papillæ discharge a granular, thickish fluid, mixed with a red powder. The mucous membrane of the pelvis is often coated with a powdery precipitate.

Under the microscope the tubules are found to contain finely granular and spherical masses (the latter provided with prolongations), which are soluble in acetic and hydrochloric acids; after a time crystals of uric acid make their appearance. They consist chiefly of urate of ammonia, in part of urate of soda.

Uric-acid infarctions in the new-born do not seem to give rise to any symptoms during life, except that a red powder remains upon the diaper. The urine often contains cylindrical bodies which are composed of spheres of urate of ammonia (vide Fig. 92). Virchow regards these infarctions as physiological, others regard them as pathological (resulting from disturbances of respiration or nutrition).

b. Lime infarctions form fine whitish streaks, which radiate from the papillæ; the looped tubes of the medullary substance are especially apt to be affected. Under the microscope, dark granules of carbonate of lime are found in the lumen, the epithelial cells, and even the membrana propria of the tubules. These infarctions are rare in childhood, more frequent in advanced life.

In a case of scarlatinous nephritis, Kuessner found deposits of phosphate of lime in the tubes of the cortex.

c. Oxalate of lime infarctions in the convoluted tubes were observed by Ko-

bert and Kuessner in rabbits which had been poisoned by oxalic acid. Fraenkel's investigations render it probable that this also occurs in human beings, and may give rise to anuria.

d. Bilirubin infarctions are found only in new-born infants who have been jaundiced. They are observed chiefly in the papillæ, rarely in the cortex. The bilirubin occurs in the form of spherical masses and irregular granules, or in the shape of needles and rhombic tablets. It is situated in the lumen of the tubes, the epithelial cells, and intertubular tissue, particularly the blood-vessels.

e. Hæmatoidin infarctions form brownish streaks in the papillæ. The hæmatoidin consists chiefly of clumps, more rarely of well developed crystals. In places we find red blood-globules, the dissolution of which has given rise to the

FIG. 93.



Casts of urate of ammonia from the urine of a new-born child suffering from uric-acid infarction. After Hofmann and Uitzmann.

formation of hæmatoidin. These infarctions are observed after transfusion of blood, extensive burns, etc.

4. *Tumors of the Renal Pelvis and Ureters.*

Cancer of the pelvis of the kidney either extends from the kidney itself, or from adjacent organs. In renal cancer separate nodules of cancer are rarely found on the mucous membrane of the lower urinary passages. Wising and Blix recently described a primary cancer of the right ureter.

Cysts are not often observed, and possess no clinical interest.

5. *Parasites in the Pelvis of the Kidney and Ureters.*

Parasites may pass from the kidneys into the pelvis and ureter, or they may have their primary seat in the latter situation. The latter include *strongylus s. eustrongylus gigas* the palisade worm and *distomum hæmatobium*.

FIG. 94

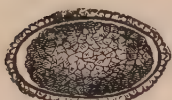


FIG. 95b.

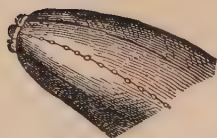


FIG. 95a.

FIG. 94.—Ovum of *strongylus gigas*. After Leuckart.FIG. 95.— *Strongylus gigas*. *a*, natural size; *b*, enlarged head with lateral papillæ.

a. Strongylus gigas is a round worm. It is apt to be mistaken for clots of blood and ascarides; it is observed most frequently in the kidney of the dog

FIG. 96.

Ova of *hematobia* in the clots of hæmaturia. Enlarged 300 times.

The symptoms are those of pyelitis (renal pain, dysuria, pyuria, hæmaturia), because the parasite irritates the mucous membrane of the renal pelvis. The diagnosis is possible if the ova are detected in the urine (vide Fig. 94).

The parasite resembles an earth worm or ascaris, but is distinguished from the latter by its red color, and by the six papillæ around the mouth. In dogs the male is 3.1 cm. long, the female 6.4 cm. The ova are elliptical, brownish, with a thick shell and round depressions on the surface.

b. *Distomum hæmatobium* is one of the leeches, and is especially frequent in Egypt. The parasite is probably swallowed in the drinking water, and then passes from the intestine into the veins of the ureter and renal pelvis. In this locality ova are deposited in large numbers, and give rise to occlusion of the blood-vessels, hemorrhages of the mucous membrane, and losses of substance. The origin of the hemorrhages may be recognized by the demonstration of the ova in the small clots of blood (vide Fig. 96). These are oval, about 0.12 mm. long, 0.04 mm. wide, and carry a prick at one end or to the side. The ova and hemorrhages may form the nucleus of a calculus.

PART IV.

DISEASES OF THE BLADDER.

A. ANATOMICALLY DEMONSTRABLE DISEASES OF THE BLADDER.

1. *Catarrhal Cystitis.*

I. ETIOLOGY.—Cystitis may be acute or chronic, primary or secondary.

Primary cystitis is infrequent and usually follows injury, chemical irritation, or colds.

The injuries include a fall or blow in the region of the bladder, the introduction of hard substances into the bladder (catheters, sounds, stone-crushers, foreign bodies). In women, injuries to the bladder followed by cystitis often take their origin in the genital apparatus (pressure of the head during delivery if the pelvis is narrow, application of the forceps, uterine pessaries). My own experience seems to indicate that obstinate constipation may act as a cause of traumatic cystitis.

Among the chemical irritants may be mentioned cantharides, the balsams, and strong acids. Individual peculiarities play an undoubted part in this respect. For example, symptoms of violent irritation of the bladder develop in some patients after the application of a single fly blister.

In some cases the chemical irritant passes through the urethra into the bladder, for example, in the careless use of urethral injections during the treatment of gonorrhœa.

This category also includes those cases in which unclean instruments and with them schizomycetes are introduced into the bladder. Musculus showed that under the influence of the bacteria conveyed into the urine, a ferment is produced which converts the urea into ammonium carbonate, renders the urine alkaline and therefore irritating, and thus gives rise to cystitis. Instruments which are passed into the bladder may thus produce cystitis in a mechanical or chemical manner. In the former event the symptoms appear almost immediately after the introduction of the instrument, in the latter event they are preceded by a stage of incubation (twenty-four to thirty-six hours), because the schizomycetes must first proliferate within the bladder.

The chemical irritants also include certain articles of food and drink, such as young beer or wine, or asparagus.

The occurrence of cystitis as the result of a cold has been denied by some writers. In some cases no cause can be ascertained.

Secondary cystitis is more frequent than the primary form. It may be the result of stasis of urine, of the spread of inflammation from adjacent organs, the sign of another affection of the bladder, or the result of infectious diseases.

When the escape of urine is impeded, and stasis occurs, the symptoms of cystitis are usually not long delayed. This takes place in urethral stricture, hypertrophy of the prostate, paralysis of the bladder, etc. The stasis itself favors the development of cystitis, but the chief factor is the alkaline decomposition of the urine, usually associated with the former. Cystitis is occasionally the result of voluntary retention of urine, particularly in females.

It may be produced by the extension of gonorrhœa, prostatitis, perimetritis, parametritis, inflammations of the uterus, ovaries, rectum, kidneys, pelvis, and ureters.

Secondary cystitis is not infrequent in other diseases of the bladder (stone, tumors, parasites).

Finally, it develops during certain infectious diseases, for example, typhoid and relapsing fever, cholera, dysentery, articular rheumatism, pyæmia, septicæmia, etc.

Cystitis is a disease of middle life and old age; in old people it is usually the result of prostatic enlargement. It is extremely rare in childhood.

II. ANATOMICAL CHANGES.—The anatomical changes vary according as the inflammation is acute or chronic, partial or total. Partial cystitis generally involves the neck of the bladder.

In *acute cystitis* the mucous membrane of the bladder presents the same changes as other inflamed mucous membranes. It is unusually red, either diffusely or in patches (the latter particularly at the top of the folds of mucous membrane). Extravasations of blood are observed here and there. The surface is coated with mucus or pus. The tissue of the mucous membrane is loosened and unusually succulent. In some cases the muscular coat is also unusually moist, thick, and brittle. Even the subserous tissue and the serous lining may be implicated in the inflammatory process (pericystitis).

Among the complications are ulcerations of the mucous membrane. These begin as epithelial desquamation, which gradually extends deeper and deeper, and may finally lead to perforation of the bladder into the peritoneum, rectum, uterus, vagina, etc.

If the inflammation is very intense, necrotic and ichorous changes sometimes develop in the mucous membrane. The latter degenerates into a pulpy, gangrenous, foul-smelling mass.

Abscesses sometimes develop in the wall of the bladder, and may rupture externally, internally, or in both directions.

In *chronic cystitis* the mucous membrane has a grayish-red, brownish-red color, or presents a pigmented, blackish, slaty appearance, as the result of previous hemorrhages and changes in the blood pigment. The veins are sinuous, dilated, and varicose in places, particularly near the neck of the bladder. The mucosa and submucosa are usually thickened. The muscular coat also is often thickened, the hypertrophy affecting partly the muscular fibres, partly the intermuscular connective tissue.

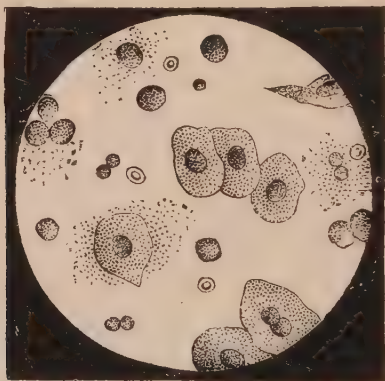
Even the serous membrane may be thickened, and the bladder is sometimes adherent to adjacent organs.

In some cases the bladder is unusually large, and extends almost to the umbilicus; in other cases it is hardly larger than an apple of moderate size. If, at the same time, the wall of the bladder is thickened, the former condition is known as eccentric hypertrophy, the latter as concentric hypertrophy. It must be remembered, however, that the contracted bladder always appears to have thickened walls. The muscular coat is often very pale and fatty.

The hypertrophic bands of muscular fibres often project in a network upon the inner surface of the bladder, the mucous membrane between the prominences being depressed.

The mucous membrane sometimes forces its way between the bundles of muscular fibres, and this results finally in the formation of diverticula. In one of my cases, the diverticulum was larger than the bladder proper.

FIG. 97.



Urinary sediment in acute cystitis, containing round cells, red blood-globules and vesical epithelium. Enlarged 275 times.

The diverticula occasionally contain calculi, and their walls are often incrustated with urates.

The remaining complications are similar to those observed in acute cystitis.

III. SYMPTOMS.—The symptoms of acute cystitis are sometimes purely local, sometimes they begin as general symptoms, or the latter are associated throughout with the former.

The disease begins occasionally with a chill or repeated chilly sensations, followed by fever, which may rise above 39° C. There is increased thirst, anorexia, general malaise, frequently, also, insomnia. The tongue is thickly coated. Nausea and vomiting, distention of the abdomen, and constipation also make their appearance.

The most important local symptom is the intolerable desire to pass water. The patients have a constant desire to micturate, but are able to void only a few drops, or none at all. Micturition is associated with violent pain, which is compared to that which would be produced by

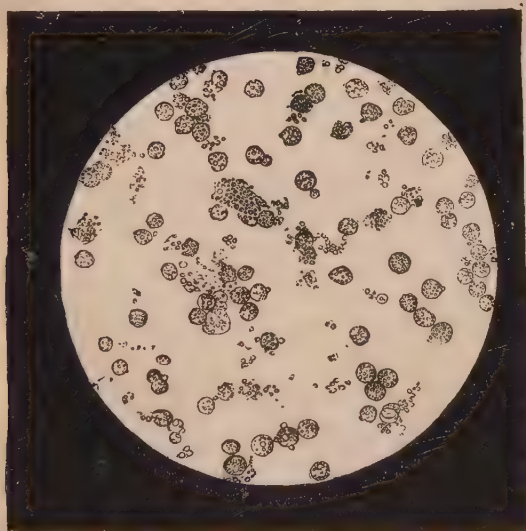
pouring molten lead through the urethra. These two symptoms are known as tenesmus of the bladder.

The tenesmus sometimes extends to the sphincter ani. The patients generally delay defecation as long as possible, because the pressure of the fæces on the bladder intensifies the pain.

There is a painful sensation in the vesical region, even during the intervals of micturition. Sometimes it is merely a dull feeling of pressure or tension, sometimes a pronounced pain, which radiates into the testicles, penis, back, or thighs. If the fundus is the chief site of inflammation, the pain is referred principally to the perineum; if the summit of the bladder is chiefly affected, the pain is most marked behind the symphysis.

The bladder is more or less tender on pressure. The introduction of the catheter is attended with great pain, and should not be performed unless absolutely necessary.

FIG. 98.



Purulent urinary sediment in acute cystitis in a man æt. 23 years. Enlarged 275 times.

At the beginning of cystitis, the urine often presents no changes, except that it becomes scanty, dark-red in color, very acid, and its specific gravity is increased. These changes may be the only ones noticed during the entire course of the disease. But, as a rule, the urine contains an unusual amount of mucus. The sediment contains more or less numerous round cells, a smaller number of red blood-globules, and often very numerous epithelium cells from the mucous membrane, which possess long prolongations when they are derived from the deeper layers of epithelium.

In some cases the urine contains blood, and a larger number of red blood-globules are then found in the sediment. In purulent catarrh there is an unusual number of round cells in the urine, and a grayish-white, purulent sediment is soon deposited at the bottom of the vessel

(vide Fig. 98). The pus-corpuscles not infrequently present amœboid movements, which may continue for more than two days (vide Fig. 99).

If the urine contains a large amount of mucus or pus, it is very apt to undergo alkaline fermentation. Disease of the bladder, therefore, may be inferred from the character of the urine only when the latter has been recently passed into a clean vessel.

In certain cases, however, alkaline fermentation of the urine takes place within the bladder. The freshly passed urine turns red litmus paper blue, and has an ammoniacal odor. If the litmus paper, which has been turned blue, is allowed to dry in the air, it gradually turns red, because the ammonia (the cause of its alkaline reaction) is volatilized.

FIG. 99.



Round cells undergoing amœboid movement in cystitis. After Michelson.

The sediment of the urine contains those constituents which are soluble only in acid fluids. These include the triple phosphates and acid urate of ammonia. The former are easily recognized by their "coffin-lid" shape; they dissolve on the addition of acetic acid. The latter, as a rule, are brownish, round bodies which possess more or less numerous prolongations (vide Fig. 100). They dissolve on the addition of hydrochloric acid and after heating. These crystals are also dissolved on the addition of potash, and bubbles of ammonia are given off at the same time. The sediment also contains carbonate and phosphate of lime. The former consists of amorphous granules or larger round bodies which not infrequently cohere into the so-called dumb-bell crystals.

These dissolve on the addition of hydrochloric acid, bubbles of carbonic acid being given off. Phosphate of lime appears in the shape of small granules or granular lumps. In addition, the sediment contains numerous rod-shaped and spherical bacteria which are in active motion.

The urine sometimes has a feculent odor. This occurs when the entire wall of the bladder has taken part in the inflammation and a diffusion of intestinal gases has taken place. The urine may also contain sulphuretted hydrogen.

If the alkaline urine also contains pus, the sediment has a ropy, gummy consistence, resembling the white of egg. This is especially marked when the urine is poured into another vessel. It may give rise to retention of urine, inasmuch as the mucous clots block the entrance to the urethra. The phenomenon depends upon the fact that the pus-corpuscles undergo a peculiar swelling under the influence of carbonate of ammonia (vide Fig. 101).

FIG. 100.



Sediment of urine, in a condition of alkaline decomposition, containing triple phosphates (coffin-lid shape), urate of ammonia and innumerable schizomycetes.

Retention of urine in cystitis is sometimes the result of nervous causes, or perhaps of marked implication of the muscular coat in the inflammatory process.

In inflammation of the neck of the bladder the pains are very severe in the perineum, the tenesmus is unusually intense, and a few drops of pure blood often make their appearance upon straining after micturition.

Suppuration of the wall of the bladder is usually attended by chills, high fever, and severe constitutional symptoms. If the abscess ruptures into the bladder, a large amount of pus suddenly appears in the urine; if it ruptures into the intestines, pus and urine appear in the fæces; rupture into the peritoneal cavity is followed by symptoms of peritonitis.

If gangrene of the mucous membrane sets in, the urine becomes blackish-brown in color, contains shreds of the desquamated tissue, and has a foul, gangrenous odor. At the same time, a low typhoid condition develops (urosepsis), and the patients often die in collapse.

Obstetricians have reported a number of cases in which the mucous membrane of the bladder was exfoliated. It occurs during pregnancy or parturition, and is usually preceded by retention of urine. Maurer has shown that severe cystitis in itself may give rise to this phenomenon. Lister reported a case of traumatic origin. Buchanan reported a case in a man *æt.* 60 years, who was relieved of a cystitis of long standing after exfoliation of the vesical mucous membrane.

The exfoliated pieces may hardly show the structure of the mucous membrane, and are incrustated with urates, phosphates, and oxalate of lime. This condition may give rise to retention of urine and severe pain.

The duration of acute cystitis varies from a few hours, as, for example, after the ingestion of injurious drinks, to several days and even weeks. The disease generally terminates in recovery, or it may pass gradually into

Fig. 101.



Sediment of urine, in a condition of alkaline decomposition (in cystitis), containing swollen round cells, crystals of triple phosphates, and urate of ammonia.

the chronic stage. Danger may arise from the development of abscess, gangrene, or retention of urine.

Chronic cystitis begins as such, or it is the result of frequently relapsing acute catarrhs. The chief symptoms are disturbances in micturition and changes in the urine.

Spontaneous pains in the region of the bladder, tenderness and vesical tenesmus are usually less pronounced than in acute cystitis. The urinary changes are similar to those observed in acute cystitis, but alkaline fermentation is more common. Retention or incontinence of urine is apt to occur. In many cases the patients pass water without much difficulty, but a certain amount always remains in the bladder. The

organ then becomes distended to such a degree that it reaches the umbilicus or even extends above it. Barkow reported a case in which the bladder contained about 4,000 ccm. of urine.

If the bladder has undergone concentric hypertrophy, the patient micturates frequently, but very little at a time. On rectal or vaginal examination, the bladder is then felt as a small, round, hard tumor.

Uræmia and ammoniæmia are more frequent than in acute cystitis. Acute exacerbations of the inflammation are very apt to develop. Many patients tolerate the disease for years, but finally grow anæmic and cachectic. Chronic cystitis may also result in the formation of calculi, or it may spread upwards to the pelvis and kidney.

IV. DIAGNOSIS.—As a rule, the diagnosis of acute and chronic cystitis is not very difficult. The prominent symptoms are disturbances of micturition, abnormal sensations in the vesical region, and changes in the urine.

With regard to alkaline urine it should be remembered that, apart from subsequent alkaline decomposition, a diagnostic mistake may arise, owing to the fact that the urine may be alkaline as soon as passed, although it has not undergone alkaline fermentation. In such cases the alkaline reaction is produced by a fixed alkali, and litmus paper, which has turned blue by the urine, will retain its color on exposure to the air. Furthermore, the sediment does not contain innumerable bacteria. The alkaline urine is passed after ingestion of large amounts of the salts of the vegetable acids, and also occasionally by patients suffering from dilatation of the stomach (vide page 86).

We must also attempt to ascertain the cause of the disease, since the treatment depends upon the etiology.

V. PROGNOSIS.—The prognosis depends mainly upon the cause of the inflammation. Acute cystitis therefore furnishes a better prognosis than chronic cystitis. It is unfavorable in cancer of the bladder and hypertrophy of the prostate.

VI. TREATMENT.—Prophylaxis is an important feature in treatment. It includes the avoidance of hurtful drinks, the rational treatment of gonorrhœa, caution in the use of cantharides, careful cleansing of all instruments which are introduced into the bladder, etc.

After the disease has developed, treatment should first be directed against the causal conditions. This includes the relief of urethral stricture, treatment of prostatic hypertrophy, vesical calculi, etc.

The symptomatic treatment of acute cystitis, is, in general, internal; that of chronic cystitis is local.

Patients suffering from acute cystitis should be kept in bed, the lower part of the abdomen constantly covered with a warm poultice, and lukewarm drinks given freely. Solid, spiced food should be avoided. The drinks may consist of tea, or, if there is an abundant secretion of mucus, an infusion of *uva ursi*. Lukewarm milk, mixed with half the quantity of lime-water, may also be recommended. Patients suffering from acute or chronic cystitis must abstain from sexual intercourse.

A daily evacuation from the bowels should be secured; if necessary, by the administration of a mild laxative.

In addition, the patient should take a warm bath (30° R.) morning and evening, remaining in the bath for half an hour.

Certain symptoms may require special treatment. If the pain in the perineum is very severe, four to six leeches may be applied in this region. In severe tenesmus of the bladder, we may introduce suppositories of opium or morphine, but should avoid the use of belladonna.

R. Opii.....gr. xv.
 Ft. c. Butyr. Cacao, q. s., supposit. No. x.
 D. S. One suppository introduced t. i. d.

R. Morphin. hydrochloric.....gr. iss.
 Ft. c. Butyr. Cacao, q. s., supposit. No. x.
 D. S. One suppository introduced two to three times daily.

Astringents and balsams may be given to diminish the excessive secretion of mucus. Among the former we may mention, in addition to uva ursi, tannic acid as one of the most certain remedies.

R. Acid. tannic.....gr. lxxv.
 Pulv. et. succ. liq., q. s. ut ft. pil. No. xxx.
 D. S. One pill taken four times a day.

Arbutin (gr. ivss.—viiss. every two hours), a glucoside obtained from uva ursi, has been warmly recommended. Our own experience with this remedy has not been very successful.

Among the balsams we may mention balsam of copaiba, Peru, tolu, turpentine (five to twenty drops in milk t. i. d.).

If irritative symptoms are not specially marked, the administration of alkaline waters is indicated. The patient may take daily from four to eight glasses of lukewarm Selters, Bilin, Vichy, Wildung water, etc. The earthy wells of Wildung and Driburg may also be recommended, if there is an active secretion of mucus.

If the urine has undergone alkaline decomposition, antiparasitic remedies are indicated, since this process is produced by low organisms. Friedreich and Fuerbringer obtained good results from salicylic acid, but in my hands this remedy has not proven very successful. Nor have I obtained very good results from the administration of potassium chlorate ($\frac{3}{4}$ ss. : $\frac{3}{4}$ x., one tablespoonful every two to three hours). Carbolic acid, benzoic acid (gr. ivss.—viiss., one powder every two hours), resorcin (gr. iss.—viiss. every two hours) and quinine have also been highly recommended.

The catheter should only be employed in cases of retention of urine.

Stimulants are indicated in gangrene of the mucous membrane of the bladder.

In chronic cystitis preference should be given to local measures of treatment.

The dietetic measures remain the same as in acute cystitis. In addition, a carefully cleaned elastic catheter should be introduced into the bladder every morning and evening, and the urine removed. The bladder should then be washed out by means of an irrigator or Hegar's funnel (vide Fig. 16, page 111), and this should be followed by the injection of medicinal substances.

If the urine is acid, or contains considerable mucus, the bladder, after being emptied of urine, should be washed with water (35° C.) until the latter runs out perfectly clear. An injection should then be made of a solution of nitrate of silver, beginning with gr. iij. : $\frac{3}{4}$ xvi., and gradually increasing to gr. xxx. : $\frac{3}{4}$ xvi. The solution should be warmed, and portions of it allowed to escape at intervals of a couple of minutes. The injection should be discontinued as soon as the patient complains of a feeling of tension in the bladder. At the same time, we should watch for the ascent of the bladder above the symphysis. We prefer a single to a double catheter.

Many other remedies have been recommended for injection of the bladder, for example :

Sodium chloride (5% solution).	
Solut. acid. tannic.	} 3 ss.-3 iiss. : $\frac{3}{4}$ xvi.
Solut. aluminis.	
Sol. zinc. sulphat.	
Sol. plumb. acetat.	
Sol. resorcin, gr. xlv.-3 iiss. : $\frac{3}{4}$ iiss.	
Sol. kali chlorat., gr. xlv. : $\frac{3}{4}$ iiss.	
Sol. zinci chlorat.	
Tinct. ferri sesquichlorat.	

When the urine is passed in a condition of alkaline fermentation, disinfectant injections are indicated. The best are salicylic acid (gr. $\frac{3}{4}$ -gr. iij. : $\frac{3}{4}$ iiss.) and salicylate of soda (gr. xv.-lxxv. : $\frac{3}{4}$ iiss.); caution must be exercised in the use of carbolic acid (gr. viiss.-xlv. : $\frac{3}{4}$ iiss.). Other remedies in this class are permanganate of potash (gr. iss.-ivss. : $\frac{3}{4}$ iiss.), resorcin, quinine.

When the disease does not yield to any form of treatment, the bladder has been opened, as in cutting for stone, emptied of its contents, and the wall of the bladder treated directly. Horwitz collected 58 cases of this kind, of which 30 were cured, and 16 died; among the latter 15 were associated with old disease of the kidney.

In eccentric hypertrophy of the bladder the urine should be withdrawn by means of the catheter three times a day, and a trial made of electrical applications and cold frictions to the region of the bladder. In concentric hypertrophy of the bladder the patient should be directed not to micturate oftener than every two hours, in order to gradually dilate the bladder.

2. Cancer of the Bladder.

I. ETIOLOGY.—Cancer of the bladder is a rare disease; among 11,811 autopsies collected by Heilbronn and Hasenclever, it occurred 77 times (0.7 per cent). Among these 77 cases, 65 occurred in women, 11 in men. It develops most frequently at the age of thirty to forty years, but in males, as a rule, it appears beyond the age of fifty years.

The disease is rarely primary. It usually extends from adjacent organs (uterus, rectum, prostate), and rarely develops as a metastasis from remote organs.

II. ANATOMICAL CHANGES.—Cancer of the bladder appears as a diffuse infiltration, in which the submucous tissue is particularly involved, as a pedunculated tumor, or as nodules which grow with a broad base from the inner surface of the bladder. The mucous membrane sometimes presents ulcerations, the edges of which are peculiarly thickened and hardened, and, under the microscope, show cancerous infiltration.

The most frequent form of cancer of the bladder is epithelioma, next villous cancer and scirrhus, finally medullary or even colloid cancer. The tumors often undergo ulceration, and may even disappear almost entirely. Gangrenous changes are also observed. The mucous membrane is generally in a catarrhal condition; calculi are often present.

The most frequent starting-points of the cancer are the fundus and neck of the bladder. It extends not infrequently to adjacent organs, and in some cases it could be followed along the urachus to the umbilicus.

Ulcerative processes often give rise to abnormal communications, or to perforation into the peritoneal cavity.

Metastases have been observed in the liver, lungs, kidneys, stomach, pleura, pericardium, peritoneum, and lymphatic glands. Marantic thrombosis develops in a number of cases. Heilbronn mentions waxy degeneration associated with cancer of the bladder.

III. SYMPTOMS.—The disease is easily recognized if we are able to demonstrate a vesical tumor, or if particles of cancer are passed in the urine.

A tumor of the bladder may be recognized by rectal or vaginal exploration, more rarely through the abdominal walls, or by combined ex-

FIG. 102.



Exfoliated villous tissue with an epithelial covering in cancer of the bladder. After Ultzmann.

amination. Upon the introduction of a catheter, we sometimes find a soft tumor within the bladder, or particles of the neoplasm remain in the fenestra of the catheter, and may be examined under the microscope. The surface of the cancer sometimes becomes incrustated with salts, so that the catheter comes in contact with a hard, scratching surface.

In rare cases, it may happen, in females, that a pedunculated cancer is forced into the urethra during micturition, and thus becomes visible.

Exfoliation of cancerous tissue and its appearance in the urine are observed particularly in villous cancer. In many cases, the vascular villi are very easily recognized (vide Fig. 102). Sometimes they have a continuous epithelial covering; sometimes this is loosened, and is found separately as a more or less coherent epithelial layer. Not infrequently

the villous tissue is necrotic, and is then recognized with difficulty. It often contains the remains of previous hemorrhages in the shape of hæmatoidin crystals, partly as rhombic tablets, partly as needles, or convoluted threads (vide Fig. 103). In other cases, the necrotic tissue contains peculiar crystals, arranged in the shape of rosettes, and which, according to Ultzmann, are probably composed of oxalate of lime. Other necrotic villi contain merely round cells, red blood-globules, and countless schizomycetes.

If the cancer is epithelial in character, the urinary sediment not infrequently contains a very large number of peculiarly shaped epithelial

FIG. 103.



Necrotic villous tissue, strewn with numerous hæmatoidin crystals, in cancer of the bladder.

cells. They are often unusually small, often multinuclear, and aggregated into coherent masses.

If a vesical tumor cannot be demonstrated, and particles of cancer are not found in the urine, the diagnosis will usually remain doubtful. In such cases, one of the chief symptoms is frequently recurring, profuse hæmaturia, especially when this occurs in old people who have a cachectic appearance and swollen lymphatic glands in the groins. It may be extremely difficult to make a differential diagnosis from hæmaturia produced by vesical calculi. In cancer, the hemorrhage often occurs while the patient is at rest; in vesical calculi, it usually follows bodily exertion.

All other symptoms are much more ambiguous. Many patients complain of pain. This is usually most violent in the perineum, because the cancer is generally situated at the fundus, but it often radiates into the testicles, penis, thighs, abdomen, and renal region. On account of the pains in the perineum, the patients often are unable to remain seated for a long time; they experience less pain on wooden benches than on soft cushions, and feel most comfortable in the horizontal position. The pains of cancer often occur during complete rest; the pains of vesical calculi develop principally after movement.

Tenesmus of the bladder appears in many cases, because, as a rule, cancer of the bladder is associated with cystitis. Retention of urine may occur from the growth of the tumor into the opening of the urethra or ureters. In the latter event, hydronephrosis will develop, and result in death from uræmia if both ureters are affected. Incontinence of urine makes its appearance if the tumor partly occludes the opening of the urethra, so that the urine may flow out constantly to one side. Priapism has been observed in some cases.

Fibrinuria has been observed in some cases of villous cancer. A few minutes after its passage, the urine is converted into a gelatinous mass, which can be gradually liquefied by shaking. The urine is occasionally a brown or blackish-green, stinking fluid.

The duration of cancer of the bladder may extend over several years, but, as a rule, the disease runs its course within a few months.

It terminates fatally in almost every case, but it is still claimed that spontaneous recovery sometimes occurs from gradual exfoliation of the cancerous tissue. Death may be the result of hemorrhage, uræmia, or increasing marasmus; it may be attended with typhoid symptoms, if the bladder becomes gangrenous, or signs of peritonitis if the cancer has extended to the peritoneum, or the bladder has undergone perforation. Secondary deposits of cancer may occupy the foreground to such an extent as to mask the vesical symptoms.

IV. DIAGNOSIS.—The disease is most apt to be mistaken for vesical calculi and simple cystitis. The prognosis is unfavorable, as in other cancers, and treatment is purely symptomatic.

APPENDIX.

Polypi, lipoma, myxoma, adenoma, papilloma, myoma, sarcoma, cysts, and cavernous angioma of the bladder have also been described. These growths possess a purely surgical interest.

3. *Parasites of the Bladder.*

Vegetable or animal parasites are found occasionally in the bladder. Innumerable schizomycetes are seen in urine which has undergone alkaline decomposition. Some individuals pass bacteria in the urine, although there is no real decomposition of the fluid (bacteruria). Such urine is generally cloudy, and not infrequently has a peculiar stale odor almost like that of meat broth. Symptoms may be entirely absent, but some patients complain of dysuria. In a case of diabetes mellitus, Kuessner found threads of leptothrix, which formed small brownish clumps in the urine. Sarcina has been observed a number of times in acid, likewise in alkaline urine. As they are usually smaller than sarcina ventriculi, they are called sarcina urinæ. The parasite is easily distinguished by its quadrilateral shape and peculiar grouping (vide Fig. 104). It is sometimes so abundant as to form a cloudy sediment. In Leube's case, the sarcina disappeared as soon as the acid urine had undergone alkaline decomposition. It has been

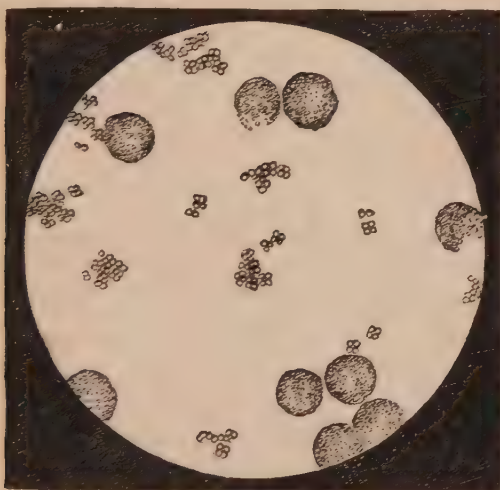
observed in Bright's disease, vesical affections, and sometimes accidentally. In the case which is illustrated in Fig. 104, it appeared in an ataxic patient, soon after a cystitis (following catheterization) had developed.

Echinococci and distomum hæmatobium have been found in the bladder. Ainsworth recently described an echinococcus of this organ. The vesicles were situated free in the cavity of the bladder; in addition, echinococci had developed in the middle lobe of the right lung.

Distomum hæmatobium is found particularly in the tropics. It has been previously mentioned that the emigration of this parasite into the vascular system of the bladder and ureters gives rise to severe inflammatory and ulcerative changes, and that the diagnosis is based on the passage of the ova in the urine (vide page 325).

As a matter of course, not all parasites, which are passed in the urine, are derived from the bladder; for example, echinococcus vesicles from the kidney

FIG. 104.



Sarcina urinae. Enlarged 750 times.

often appear in the urine. In certain cases, intestinal parasites pass into the bladder through an opening between the two viscera.

4. Foreign Bodies in the Bladder.

We will not consider those foreign bodies which are introduced into the bladder through the urethra, since they belong to the domain of surgery. Foreign bodies may also enter the bladder when this organ communicates with other abdominal viscera. In many cases, the passage of vegetable cells or striated muscular fibres in the urine has proven the existence of a vesico-intestinal fistula. The passage of flatus through the urethra has also been observed in a few cases.

Gall-stones have been found occasionally in the bladder, evidently as the result of previous ulceration of the biliary passages into the bladder.

The passage of hair in the urine (pilimictio s. mictus pilum) has been reported in a few cases. This is sometimes owing to voluntary or involuntary deception (hysterical females). In a number of cases, the bladder communicated with a dermoid or ovarian cyst, so that hairs, cheesy matter, even bones and teeth, have been discharged in the urine. Whether true trichiasis vesicæ—the growth of hair upon the vesical mucous membrane—really occurs is more than doubtful. Martini recently described a case in a new-born child. There was atresia of the urethra and anus, and the descending colon was connected with the bladder. The posterior portion of the bladder presented the microscopic appearances of the

external integument, and was covered with fine hairs. Martini believes that this was not an instance of true trichiasis, but an inclusion of embryonal germs.

B. FUNCTIONAL DISEASES (NEUROSES) OF THE BLADDER.

1. *Nocturnal Enuresis.*

1. Nocturnal enuresis is almost exclusively a disease of childhood. The patients micturate unconsciously during profound sleep, usually during the first two hours of sleep, more rarely in the early morning hours. It occurs generally in children who have a very sound sleep. The patients often state that the micturition is accompanied by dreams of standing near a brook, sitting on the vessel, etc. The amount of urine discharged is sometimes astonishing. In almost all cases, micturition is performed only once during the night.

Remissions and exacerbations of the disease frequently occur. The patients remain entirely free from it for weeks and months, and the hope of permanent recovery is entertained, when a period arrives during which enuresis occurs nightly. Remissions are observed more frequently in summer than in winter.

The children sometimes manifest a shy, irritable disposition, probably because they are usually the butt of those around them. In addition, they are sometimes very pale, and appetite is either lost, or is insatiable. If the disease has lasted for a long time, involuntary micturition may occur during the day, as the result of bodily or mental exertion. Indeed, it may finally terminate in constant incontinence. But fortunately such cases constitute rare exceptions. As a rule, the disease appears from the age of 3 to 12 years, after which it ceases spontaneously, sometimes even suddenly.

2. The causes vary greatly, and we should endeavor to determine the etiology in each individual case.

In certain cases, it is the result of bad training. It is well known that children under the age of 2 years usually pass urine and feces in the clothing. The ability to control the sphincters of the bladder and rectum is acquired gradually. This is secured most readily by seating the children on the vessel at certain definite intervals, and then allowing them to satisfy their natural wants. If this habit is neglected, particularly at night, nocturnal enuresis not infrequently develops.

The disease results occasionally from dietetic errors. It is observed in children who eat heartily, especially of fluid food, shortly before going to bed. Fruit and poor beer seem to be especially injurious. Distention of the bladder or an irritating condition of the urine may then be regarded as the real cause of the disease.

It is observed at times in children who sleep under heavy feather beds. The children are awakened at night by the desire to urinate, but feel too comfortable to lift the warm bed covering and use the vessel. The bladder is then distended, and involuntary micturition subsequently occurs during sleep.

Some cases are the result of conditions of irritation in or near the urinary passages. Thus the affection is observed in girls who suffer from small polypoid excrescences near the urethra. Removal of the growths with the scissors cures the disease. Enuresis may also develop in children who suffer from pyelitis or vesical calculi, especially when the latter are situated near the neck of the bladder.

The irritation produced by worms may also give rise to enuresis; in two of our cases, the affection ceased at once after oxyurides had been evacuated.

Constitutional changes must also be taken into consideration. More convincing proof is necessary, it is true, in order to show that enuresis nocturna is especially frequent in scrofulous and rachitic children, or in those whose parents have suffered from arthritis and epilepsy, but we cannot resist the impression that the disease is frequent in very pale and nervous children. Epilepsy may also appear in the form of nocturnal incontinence. It is occasionally the result of masturbation.

We may also mention that it appears occasionally in adults as the precursor of grave cerebral and spinal diseases.

Whether the disease is more frequent in boys than in girls, as is often asserted, has not been positively determined. It has sometimes been observed in the form of an epidemic in large orphan asylums. When constitutional factors enter into the etiology, it depends evidently on disturbances of innervation, but whether the detrusor or sphincter vesicæ is most affected or whether it is the result of sensory disturbances is unknown.

3. Treatment depends, as a matter of course, upon the etiology, but the following four rules hold good in all cases: *a.* The patient should empty the bladder night and day at fixed intervals, and should be roused at night for this purpose. *b.* Supper should be taken at least an hour before going to bed; distention of the stomach, spicy food, and fluids should be avoided. *c.* The mattress should be hard and the bed-clothes thin. *d.* The patient should assume lateral decubitus.

Medicinal treatment will hardly prove successful unless the above-mentioned rules are carried out. If the patient is pale, we may order iron preparations, cold frictions, douches, baths, or a trip to the mountains. If epilepsy is hereditary in the family, we may prescribe potassium bromide ($\frac{3}{4}$ ss. : vii., one tablespoonful t. i. d.). Considerable use is made of narcotics: atropine, ext. belladonnæ, chloral hydrate, nuxvomica, ergotin, etc. A fly blister to the spine and tincture of cantharides internally have also been recommended.

The mechanical treatment includes: introduction of bougies, cauterization of the urethra and neck of the bladder, the application of compressors which obstruct the escape of urine and thus waken the patient, the application of collodion to the meatus of the urethra or prepuce.

Great benefit may be derived from the application of the faradic current. Ultzmann lays stress on the synergy of the sphincter ani and sphincter vesicæ, and therefore recommends that one electrode be introduced into the rectum, the other applied to the perineum, or in girls, to a gluteal fold. The sittings should last five to ten minutes, and be held every other day. Selligmueller introduces a brass rod about two cm. into the urethra, and connects it with the cathode of a secondary faradic current, while the anode is placed above the symphysis; current barely perceptible, duration of each sitting five minutes. Erb also recommends galvanization of the lumbar cord and of the spinal cord in general.

2. *Hyperæsthesia of the Bladder.*

This affection is characterized by the fact that a small amount of urine in the bladder gives rise to the desire to urinate. The patients sometimes empty the bladder more than four times in a single hour. As a rule, micturition is unimpeded, and the urine is unchanged, or, in certain cases, it is very pale or unusually acid.

This is observed most frequently in adults, occasionally in children during the period of dentition. In certain cases it is the result of a bad habit, and can be overcome by directing the patient to retain the urine as long as possible. In other cases it occurs in anæmic, hypochondriacal, or hysterical individuals (disturbance of innervation), and is observed often in masturbators and those who indulge excessively in sexual intercourse. Under such circumstances, the treatment includes the administration of preparations of iron, potassium bromide, belladonna, cold baths, a trip to the country, and cold-water cures.

3. *Spasm of the Bladder. Cystospasmus.*

1. Spasmodic conditions of the muscular structure of the bladder may affect the detrusor or sphincter vesicæ or the entire muscular coat. We do not refer now to those cases in which the disease is produced by anatomical changes in the bladder (inflammation, cancer, calculi, etc.), but only to those which constitute an independent neurosis, *i. e.*, one independent of anatomical changes.

This neurosis occurs most frequently in women during middle life, especially in anæmic, nervous, and hysterical individuals. In some cases it is secondary to grave diseases of the brain or spinal cord, and also appears in masturbators and those who indulge excessively in sexual intercourse. Cystospasmus also occurs in those who have suffered from gonorrhœa or gleet. Finally, it may be a reflex effect of diseases of the uterus, ovaries, and rectum, or even of constipation or intestinal worms.

2. If the detrusor vesicæ is alone affected, the patients suffer constantly from a desire to urinate; this may be unattended with pain. The patient often micturates every ten minutes, sometimes there is constant incontinence (diuresis spastica). The urine is generally very clear, feebly acid or neutral, rarely alkaline, of low specific gravity, and increased quantity (urina nervosa *s.* spastica). The condition might be mistaken for hyperæsthesia of the bladder, were it not for the fact that it occurs in spasmodic attacks, so that remissions and exacerbations alternate with one another. The individual attacks occur spontaneously or as the result of mental excitement, occasionally of a cold. It sometimes lasts hardly longer than half an hour, but may recur many times a day, and occasionally for weeks and months.

When the sphincter vesicæ is affected by the spasm, the act of micturition is interfered with. Despite the desire to urinate, the urine is voided in a feeble stream or appears in drops, dysuria spastica, or there is complete retention of urine (ischuria spastica). At the same time the patients are annoyed by severe pain, which is apt to increase in intensity towards the end of micturition. The pain often radiates into the penis, testicles, or perineum and is associated with tenesmus of the sphincter ani.

The pains are increased to a still greater degree if the detrusor and sphincter vesicæ are affected at the same time. Spasm of the former

muscle gives rise to the desire to urinate, but this is prevented more or less completely by the spasm of the sphincter. The severity of the pain makes the patient grow pale, the skin becomes cool, drops of perspiration appear upon the face, the pulse becomes small and frequent, the entire body trembles, and sometimes syncope or general convulsions are observed.

3. The disease is easily recognized, but we should never fail to make a careful examination of the bladder with the sound, or by the introduction of the finger into the vagina or rectum, in order to exclude anatomical lesions.

4. To remove the causes of the disease, we may employ cathartics, anthelmintics, preparations of iron, nervines, etc.

In order to relieve the attack itself, the patient should be placed in a warm bath (30° R.) and directed to pass water in the bath. After this the region of the bladder and the perineum should be covered with a warm poultice, a subcutaneous injection of warm water made in this region, or suppositories of opium or morphine introduced into the vagina or rectum, or an enema of chloral hydrate given, or Dover's powder internally. If the dysuria or ischuria persists, a flexible catheter, which has been smeared with morphine ointment, may be cautiously introduced into the bladder. In obstinate cases we may employ electricity: galvanic current, cathode to the symphysis, anode to the sacrum or perineum.

4. *Paralysis of the Bladder. Cystoplegia.*

1. Paralysis of the muscular structure of the bladder may affect the detrusor or sphincter vesicæ, or both; the paralysis may be complete or partial.

Some cases result from diseases of the central nervous system. Thus, paralysis of the bladder is a very common symptom of locomotor ataxia. In certain cases it begins very suddenly, for example, after concussion of the spinal cord by a fall or blow. It is also observed in hysteria.

Vesical paralysis may occur in severe affections of the sensorium, as in cerebral hemorrhages, meningitis, severe infectious diseases, high fever, etc.

Some time ago I was called to see a young lady who had begun to complain of headache eight hours previously, and had been comatose for four or five hours. On examination, I found the bladder projecting three fingers' breadths above the umbilicus, although the patient had passed water before the occurrence of coma. As there had been no previous disturbances of micturition, it was concluded that the patient was suffering from a disease which gives rise to an unusual secretion of urine (diabetes mellitus), and that the condition was one of diabetic coma. Examination of the urine verified the suspicion. Death occurred in twelve hours.

Paralysis of the bladder is sometimes associated with marasmus, for example, in enfeebled old people, in convalescence from severe diseases, and in typhoid fever (probably as the result of anatomical changes in the muscular fibres of the bladder). It is not infrequent in onanists and those who lead a dissipated life.

Toxic paralysis of the bladder is observed in opium poisoning.

Certain cases are the result of a local affection of the bladder. Thus, paralytic conditions of the bladder are observed in individuals who have purposely retained the urine too long; this is noticed particularly in

women, artists, orators, etc. A fall or blow in the region of the bladder or severe compression during difficult labor may also give rise to paralysis of the bladder. In certain cases, violent cystitis extends to the muscular coat of the bladder, and renders it parietic. Finally, the disease develops not infrequently in individuals suffering from urethral stricture, or prostatic hypertrophy, because these conditions give rise to retention of urine and distention of the bladder.

2. In paralysis of the detrusor vesicæ the desire to urinate is felt very rarely. In some cases the bladder forms a tumor which extends to the ensiform process, and has been mistaken for pregnancy, ovarian tumors, etc. The urine is not discharged in a vigorous curved stream, but falls vertically from the meatus of the urethra. The patients often assume a bent-over-forward position, take a deep inspiration and hold it, and then employ the abdominal muscles in order to empty the bladder as thoroughly as possible. They sometimes attempt to facilitate micturition by stroking the region of the bladder. If the catheter is introduced, after micturition is completed, a certain amount of urine will be evacuated, the quantity varying according to the degree of paralysis.

Paralysis of the sphincter vesicæ causes incontinence of urine, but this form of paralysis rarely occurs separately. If the muscle is merely parietic, the patients must micturate more frequently, because the sphincter offers very little resistance to the accumulation of urine in the bladder, and to the power of the detrusor. In addition, the patients are apt to wet their clothes before they reach the closet.

There is often a combined paralysis of the detrusor and sphincter, and the symptoms mentioned above are then associated with each other. The bladder is excessively distended, and runs over as soon as its contents exceed a certain amount. ♦

3. The diagnosis of paralysis of the bladder is easy. Pain is absent, unlike inflammatory and spasmodic changes. The absence of phimosis, urethral stricture, and hypertrophy of the prostate readily distinguishes the disease from mechanical obstruction to the escape of urine.

4. The prognosis depends upon the causes. It is unfavorable when it is the result of diseases of the central nervous system.

5. The treatment is local and general. If the detrusor is paralyzed, and there is an excessive accumulation of urine in the bladder, the catheter should be introduced several times a day. If the accumulation of urine is very large, it should not be withdrawn at once, because sudden syncope and even death have been observed under such circumstances. Individuals who suffer from paralysis of the sphincter and incontinence of the urine should wear a urinal.

The introduction of the catheter mechanically irritates the muscular coat of the bladder and stimulates it to contraction. Pitha recommends that a rubber bougie be introduced as far as the entrance of the bladder, and allowed to remain there a few minutes until the patient feels the desire to urinate.

The following measures have been recommended to restore the contractility of the over-distended bladder: cold injections into the organ, cold frictions over the region of the bladder and the small of the back, cold douches, cold-water cures, sea-baths, injections of strychnine or ergotine in the region of the bladder. In paralysis of the detrusor, Erb recommends the application of the anode to the lumbar spine, the cathode to the symphysis; in paralysis of the sphincter and incontinence, the cathode should be placed on the perineum. If both muscles are

paralyzed, both methods should be adopted in succession; moderately strong galvanic current, duration of each sitting five to ten minutes. The same rules hold good with regard to the application of the faradic current. Catheter-shaped electrodes may also be employed and introduced into the bladder, vagina, or rectum. The external electrode is applied as in percutaneous electrization.

The general treatment depends upon the etiology.

PART V.

DISEASES OF THE MALE SEXUAL APPARATUS.

1. *Impotence.*

Impotence is the inability to perform the sexual act. It may be the result of local diseases of the sexual organs, constitutional diseases, or psychical influences; in rare cases, toxic influences play a part in the disease. The condition may be congenital or acquired, temporary or permanent.

Among diseases of the sexual organs, congenital or acquired changes in the penis must first be considered. In certain cases the organ is so short that it cannot enter the vagina, even when erect. The penis is occasionally shortened by extensive hydrocele, or a large inguinal hernia, inasmuch as its external integument is employed to cover the abnormalities referred to. In such cases, the impotence is temporary, and the ability to copulate returns as soon as the causative morbid conditions are removed. In certain cases there are abnormal flexions of the penis which render its introduction into the vagina impossible. These may be congenital (abnormal development of folds, or excessively short frænum), or acquired (injury, extravasations of blood and callous cicatrices in the corpora cavernosa). Ossification and tumors of the penis may act in a similar manner.

Diseases of the testicles give rise to impotence if they interfere with the production of semen. Such conditions may also be congenital (defective development of the testicles) or acquired (destruction of the testicles by inflammation or tumors). In such cases, impotence is usually the result of the absence of erections.

Similar conditions may result in consequence of certain constitutional diseases. Thus, sexual desire and the ability to obtain erections of the penis are often lost in locomotor ataxia and diabetes mellitus, sometimes after periods of unusual sexual excitement. This may also happen after chronic diseases of the digestive organs, chronic renal diseases, prolonged losses of vital fluids, prolonged onanism or other sexual excesses.

Psychical impotence is an important form of the disease. Sexual desire is sometimes abolished, and the erections of the penis remain absent or imperfect as the result of fear or worry. The first attempt at coitus often fails, because the erection is either entirely absent, or subsides too quickly, or because the emission of semen takes place before the penis enters the vagina. Then is often added the further factor of fear that renewed attempts will also prove unsuccessful, and this very fear may be the cause of continued impotence. It is noteworthy that this psychical impotence is sometimes manifested only towards certain women. These patients sometimes resort to very disgusting manœuvres in order to secure erections and the power of copulation.

It is said that bromide of potassium, camphor, lupulin, arsenic, salicylic acid, or morphine may produce impotence.

The prognosis depends mainly on the causes of the condition. If these cannot be removed, the impotence remains permanent.

Operative interference may prove necessary, if there are mechanical

obstacles to the introduction of the penis into the vagina. If the impotence is the result of constitutional diseases, treatment must be directed towards the primary affection. In psychical impotence, the best results are obtained by cheering words of advice. The patient should be told that the condition is a frequent one, and is often overcome. If the constitution of the patient is enfeebled by sexual excesses, we should recommend moderation in venere, prescribe iron preparations, a trip to the country, the use of cold frictions, douches, and cold-water cures. The galvanic and faradic currents have been successfully employed in many cases.

2. *Sterility in the Male. Aspermatism and Azoospermia.*

The term sterility in the male is applied to conditions in which, despite the ability to copulate, the female remains barren, either because the semen does not enter the female genitals during copulation, or because it is destitute of the fructifying elements, the spermatozoa. The former condition is known as aspermatism or aspermism, the latter as azoospermia or spermatozia.

a. Aspermatism is produced when there are obstructions to the permeability of the ejaculatory ducts or urethra. If the obstruction is situated in the vasa deferentia, azoospermia is produced, because the secretion of the seminal vesicles and prostate may be ejaculated during coitus. The causes of aspermatism may be congenital or acquired, organic or psychical.

Acquired aspermatism is the more frequent form, and is especially apt to follow gonorrhœa, if this has given rise to stricture of the urethra or inflammatory swelling of the prostate and compression of the ejaculatory ducts.

It is sometimes found that ejaculation of semen from the meatus of the urethra does not take place until after coitus is completed and the penis has become flaccid, because erection has produced such an effect upon the shape of the strictured portion that the semen could not escape during that period.

In some cases phimosis may give rise to aspermatism. Amussat has recently reported a case of this kind.

Diseases of the prostate may also act as etiological factors. This may happen in marked enlargement of the prostate and compression of the ejaculatory ducts, in retraction and atrophy of the gland; the ejaculatory ducts sometimes remain permeable, but prostatic changes give them an abnormal direction, so that the semen is emptied into the bladder. Under such circumstances ejaculation of semen does not occur during coitus, but after a time the semen is discharged in the urine.

In a few cases diseases of the seminal vesicles produce aspermatism. This category includes the formation of concretions which compress the ejaculatory ducts. After aspermatism has lasted for some time, a painful ejaculation of semen occasionally takes place suddenly during coitus, apparently because the obstruction has been removed. The semen has also been observed to contain blood, pus, or concretions.

Injuries sometimes act as a cause. Thus, injury to the perineum may lead to aspermatism by producing pressure on the ejaculatory ducts. It has also been observed after lateral lithotomy, because the ejaculatory ducts were injured and, later, became obliterated. Foevan states that this danger is so much greater if the stone is large and the incision

small, since rents in the incision are apt to be produced, under such circumstances, during extraction.

Psychical causes may also produce aspermatism. Coitus is then performed normally, except that ejaculation remains absent. As a rule, this occurs in nervous individuals or in those who had formerly masturbated or indulged in sexual excesses. Ejaculation sometimes occurs only during intercourse with certain women.

Finally, there are certain cases in which the ability to copulate is retained, but the ejaculation remains absent, although pollutions, with the characteristic sensations, often occur during sleep. This is probably the result of unknown congenital conditions of disturbed innervation.

The diagnosis is easy from the history of the case, but we should also endeavor to ascertain the etiology, upon which the prognosis and treatment depend. In some cases surgical interference is necessary (relief of phimosis, urethral stricture, etc.). In others we must rely on preparations of iron, douches, cold baths, electricity (psychical aspermatism). In many cases treatment is entirely useless.

b. Azoospermia, i. e., the absence of spermatozoa in the semen, will develop if the tissue of the testicles is disorganized to such an extent that spermatozoa are no longer produced, or if the testicles furnish a normal secretion, but the latter does not enter the ejaculatory ducts, on account of disease of the epididymes or vasa deferentia. The former variety is now commonly excluded from the category of azoospermia, and this appears so much the more justifiable in view of the fact that, in the majority of cases, it is combined very soon with impotence. The latter variety is generally the result of gonorrhœa, more rarely of injuries, if these have given rise to inflammation, compression and obliteration of the epididymes or vasa deferentia. This condition is rare because it is not produced unless the organs are affected on both sides. The ejaculated fluid may have the smell of semen, because this is derived from the prostatic secretion.

Azoospermia is said to occur occasionally in healthy men who present no abnormalities of the sexual apparatus.

Very little can be effected by treatment. Spontaneous recovery has been observed in a number of cases.

3. *Involuntary Discharge of Semen. Spermatorrhœa.*

In spermatorrhœa the secretion of the sexual glands escapes under other conditions than during coitus. We must distinguish true spermatorrhœa, *i. e.,* the discharge of the secretion of the testicles, and discharges from the prostate (prostatorrhœa), the seminal vesicles, or Cowper's and Littre's glands.

a. True spermatorrhœa may appear as nocturnal pollutions. These occur from time to time after the age of puberty has been reached; the individual has voluptuous dreams, the penis becomes erect, and the semen is discharged with the characteristic sensations. The pollution is followed by no bad effects; indeed, many individuals feel better, fresher afterwards. Probably an excessive accumulation of semen in the sexual glands, particularly the seminal vesicles, causes excessive tension of the walls of these structures, and this, in a reflex manner, gives rise to the discharge of semen.

Some healthy individuals have only a single pollution in one or two months, others may have one or two a week. As a general thing, they

occur so much more frequently the more vigorous and continent the individual.

The condition becomes morbid, if the pollutions occur too often, are associated with abnormal symptoms, and are followed by injurious effects. The pollutions may occur one or more times a night, and the erection of the penis may be imperfect or absent, the ejaculation may take place without erotic dreams and the characteristic sensations, and may be followed by a feeling of fatigue, restlessness, etc.

After this has lasted for some time, pollutions may also occur during the day. The discharge of semen at first takes place only during psychical or bodily excitement, later on reading obscene books, touching or even looking at a woman. The accidental contact of the penis with the clothing sometimes is followed by an emission. Under such circumstances erection is usually imperfect or absent, and the characteristic erotic sensations are also wanting.

In the most severe form of spermatorrhœa, semen is constantly

FIG. 105.



Secretion in true spermatorrhœa. The spermatozoa partly undeveloped. Enlarged 275 times.

secreted, the penis being flaccid, and voluptuous sensations absent. Such cases are infrequent, but six have come under our observation. In three, the semen appeared normal; in a fourth it contained yellowish, opaque, puriform streaks. The latter case occurred in a man of sixty-three years, who was a confirmed onanist to the last, and had never had sexual intercourse. In one case a large part of the spermatozoa were not fully developed; the head being covered with a sort of cap, which was slit in some, and hung down along the side of the neck (vide Fig. 105). Other authors mention defective development of the caudal end, marked brittleness, and diminished or absent movements of the spermatozoa. So-called semen cells, *i. e.*, large cells with five to twelve nuclei, have also been observed. The spermatorrhœa may be so profuse that the præputial sac contains a considerable amount of semen, and when this is cleaned the seminal fluid at once begins to ooze. I have repeatedly observed that an abundant admixture of semen in the urine has given rise to lipuria, so that large drops of fat floated upon the surface of the urine. In one of Frerichs' cases the urine had a chylous appearance, and contained a remarkable amount of semen.

Morbid losses of semen often exert an injurious effect upon the mental and bodily functions, though these effects are grossly exaggerated in the descriptions furnished by the books placed by charlatans in the hands of the laity. Some patients have a pale, greenish-yellow, hollow-eyed appearance. They emaciate despite the fact that astonishing amounts of food are sometimes ingested, lose muscular vigor and endurance, have a tired, dragging, even tottering gait. Many complain of abnormal sensations—formication and coldness of the limbs, stiffness and pain in the spine. The patients sometimes complain of ringing in the ear and difficulty of hearing, at times of impaired vision. According to some of the older writers, even amaurosis may occur. There is often a feeling of mental confusion, pain in the head, and dizziness. The appetite varies, in some there is insatiable hunger, in others complete anorexia. There is often obstinate constipation, sometimes frequent desire to urinate.

Many patients complain of palpitation, dyspnoea, a feeling of oppression and throbbing in the head, particularly when brought in contact with strangers. Such patients become shy and morose, often apathetic. Epilepsy, psychopathy, and locomotor ataxia have been attributed by some writers to spermatorrhoea.

Onanism is the most frequent cause of the disease. The abnormally violent and frequent irritation to which the sexual organs are thus subjected gives rise gradually to hyper-irritability and finally relaxation of the genitals, so that at first slight irritants give rise to a discharge of semen, later its escape becomes constant. Similar conditions may also develop from excesses in venery.

Local diseases of the genitals or adjacent organs occasionally act as etiological factors. They may give rise to onanism or may directly irritate the genitals and cause the discharge of semen. This category includes phimosis, retention of preputial sebum and calculi, gonorrhoea, inflammation and irritation of the prostate and seminal vesicles, vesical calculi and inflammation, helminthes, hæmorrhoids, fissure and eczema of the anus, congenitally short frænum præputii, etc.

Spermatorrhoea is sometimes the result of diseases of the brain and spinal cord. It is well known that abnormally frequent pollutions may occur at the beginning of locomotor ataxia. Fuerbringer recently described a case of fracture of the spine with injury of the dorsal cord in a man of 68 years in whom constant spermatorrhoea developed, the penis being in a condition of semi-erection. Spermatorrhoea has also been observed after epileptic seizures.

It may also appear in constitutional diseases, such as diabetes mellitus, phthisis pulmonum, and convalescence after typhoid fever or small-pox. (Gueterbock noticed post-mortem discharges of semen in the bodies of cholera patients.)

In some cases there appears to be a congenital and hereditary predisposition to the disease. It occurs in individuals who belong to a nervous or psychopathic family, and who have been unusually excitable since youth. According to Trousseau, children who suffer from nocturnal enuresis are apt to develop spermatorrhoea in later life.

The diagnosis is easy if the microscope is employed. As a matter of course, however, the presence of spermatozoa in the urine is not positive evidence of spermatorrhoea, since they may be contained in the first portions of urine passed after normal pollutions or coitus. We should always endeavor to ascertain the cause of the disease. Onanists often

deny their vice, but it can often be recognized from the appearance and demeanor of the patient. The prognosis always depends upon the causation.

Treatment must take into consideration the etiological conditions. In addition, stress must be laid on general treatment: nourishing food, daily evacuations from the bowels, avoidance of hearty meals or stimulating drinks at night. The patients should assume lateral decubitus while asleep. Feeble individuals may be treated with tonics, sea-bathing, cold-water treatment, douches. Potassium bromide (gr. xlv.-lxxv. pro die) camphor, lupulin (gr. ivss. four times a day) have been recommended in very excitable individuals. If the genital organs are flaccid, we may order strychnine and the galvanic and faradic currents.

b. Prostatorrhœa is the involuntary discharge of prostatic secretion from the urethra. This is noticed in irritative conditions of the pro-

FIG. 106.



Secretion in prostatorrhœa, after the addition of phosphate of ammonia; it contains sperm crystals, amyloid corpuscles, round and epithelium cells. After Fuerbringer.

state, whether produced by gonorrhœa, onanism, or senile hyperplastic changes. The secretion often appears while the patient is straining at stool, or while coughing, and it may often be discharged by pressing the finger, which has been introduced in the rectum, against the prostate. The fluid is generally thin, milky, and has the characteristic seminal odor. The cellular elements are cylindrical epithelial cells, round cells, laminated amyloid bodies, shining granules, and yellow pigment in flakes or granules. If a one-per-cent solution of ammonium phosphate is added to the microscopical preparation, the so-called sperma crystals, first described by Boettcher, are precipitated after a while in the shape of double pyramids or rosettes (vide Fig. 106). These are identical with the Charcot-Neumann-Leyden asthma crystals. In addition, coffin-lid crystals of triple phosphates make their appearance.

c. Secretion from the seminal vesicles is characterized by the fact that it contains gelatinous bodies which may attain the size of a pea and resemble swollen sago grains (so-called Lallemand-Trousseau bodies). According to Fuerbringer, they are composed of a globulin substance.

d. Finally, there are discharges from Cowper's glands, and probably also from Littré's urethral glands. These include those drops of viscid fluid which usually collect between the lips of the meatus urethræ after prolonged erections. The fluid is odorless, clear, stringy, contains mucus, and, under the microscope, epithelium and round cells. This condition is not serious, and requires no special treatment.

APPENDIX.

DISEASES OF THE SUPRARENAL CAPSULES.

Bronzed Skin. Addison's Disease.

I. ETIOLOGY.—In 1855, Thomas Addison first called attention to the fact that diseases of the suprarenal capsules give rise to a peculiar group of symptoms, among which changes in the color of the skin and increasing weakness play a prominent part. In 1869, Averbeck collated 126 cases, and in 1875 Greenhow furnished statistics embracing 330 cases.

It must be kept in mind that Addison's disease is not a morbid entity, and that various abnormal conditions in the suprarenal capsules may give rise to its clinical symptoms.

The disease occurs particularly from the age of 15 to 40 years. It has not been observed beyond the age of 60 years, nor is it frequent in childhood. Among 290 cases Monti found only 11 in children; one of these cases occurred at the age of three years, another at 11 years, the others at a later period.

Among 183 typical cases collected by Greenhow, 119 occurred in men, 64 in women.

Social standing exercises an etiological influence, since experience teaches that the disease is more common among the poorer classes. The influence of heredity has not been demonstrated.

In some cases the disease is primary, and the suprarenal capsules are the only organs affected. Injury in the renal region has sometimes been mentioned as the exciting cause, and likewise worry and depressing emotions. Not infrequently no cause can be discovered.

Disease of the suprarenal capsules and bronzed skin develop secondarily in connection with pulmonary phthisis, tuberculosis of the urogenital apparatus, tuberculosis of the spine, gastritis and enteritis, cancer of various organs, and marantic conditions in general, if they predispose to waxy degeneration.

II. SYMPTOMS.—The symptoms of Addison's disease are almost always preceded, for a longer or shorter period, by prodromata. In certain cases these may last many months. They consist mainly of disturbances of digestion, an increasing feeling of weakness, and profound depression.

The patients lose appetite, suffer from nausea, vomiting, and eructations, complain of pain and fulness in the epigastrium, and not infrequently suffer from obstinate diarrhœa. They emaciate more and more; feel weak, must keep to bed for days and weeks, and not infrequently fall into a desperate frame of mind. In many there is a sensation of

pressure or pain in the loins; there are also frequent complaints of rheumatoid pains in the muscles, but especially in the joints.

The clinical history becomes clearer as soon as tegumentary changes make their appearance. So long as these are absent, the diagnosis must usually be held in abeyance. The changes first appear in those places which are exposed to the air: the forehead, cheeks, dorsum of the hand, forearms, dorsum of the foot, and legs. Next in those places which are characterized normally by an abundance of pigment: the nipples, axillæ, or external genitals. This is often followed by changes in those localities in which the skin is subject to pressure or friction, such as the inner surface of the thighs, near the knees (pressure of garters); finally, the entire integument is involved.

The disease is manifested by an unusual abundance of pigment in the skin. At first the skin assumes a light-gray or smoky gray color, so that it looks as if it were not kept clean. The dark color appears in irregular patches which gradually fade into the surrounding integument. The most recent patches are hardly larger than a pea. The more extensive changes result partly from peripheral growth, partly from coalescence of individual patches.

The more the tegumentary changes advance, the darker the color of the skin becomes. It looks like graphite, or the color of a mulatto, and finally presents a bronzed appearance. The patients often present an appearance as if painted in sepia.

The palms of the hands and soles of the feet usually remain free from pigmentation, at the most they contain a few patches.

Even after the integument is diffusely pigmented, individual parts have a more intense color, so that the skin appears speckled in places. An unusual development of dark hairs has been noticed, in a few cases, in the darker portions of the skin. Tegumentary cicatrices sometimes remain entirely free from pigmentation, sometimes they are pigmented with unusual intensity. A peculiar exhalation from the skin is occasionally noticeable, and Greenhow reports a case in which a cadaverous odor was emitted for several days before death.

The sclera has a brilliant white color; the teeth are sometimes unusually clear and white; the nails of the fingers and toes may also present a remarkably white color.

Patches of pigmentation may also appear upon the mucous membrane of the mouth and pharynx, more rarely upon that of the genitals. These patches usually have a more intensely black color, are more sharply defined, and the boundaries are not infrequently jagged and very irregular. This is observed most frequently on the mucous membrane of the cheeks, sometimes in parts which are in contact with the teeth. The conjunctiva remains unaffected in almost all cases, but in a case described by Gerhardt, there was pigmentation of the rim of the ocular conjunctiva.

Other tegumentary changes have also been described in a few cases, viz., sclerodermia, vitiligo, and area Celsi.

With the increase in the tegumentary changes, the digestive disturbances and feebleness assume the upper hand. Almost uncontrollable vomiting and diarrhœa occur for days and weeks in some patients. As a rule, there is complete anorexia, but Kussmaul noticed boulimia in one case. Increased thirst has been observed in a few cases. Towards the end of life, sprue may develop in the buccal cavity.

The amount of urine is generally diminished, but Gerhardt and Wilks have observed polyuria. Its color is generally dark. In two cases, Rosenstein found diminution in the amount of urea and increase of indican; the latter was not observed in four of my cases. Thudichum noticed increase of one of the urinary pigments (uromelanin). Gerhardt and Reichardt found traces of taurocholic acid and a large amount of fatty acids in the urine. Albuminuria occurs not infrequently towards the end of life, more rarely it is present from the beginning.

Rockwell mentions rapid abolition of sexual desire as a symptom.

The increasing weakness is manifested by the subjective sensations, the inability to remain on the feet for any length of time, attacks of syncope, and certain symptoms on the part of the circulatory apparatus.

As a rule, the pulse is very rapid, but small and soft. Anæmic systolic murmurs are heard not infrequently over the heart. I have also been able to demonstrate dilatation of the right ventricle in some cases. The carotids sometimes pulsate very vigorously, and a cardiac systolic murmur is heard over them; a cardiac systolic arterial sound is heard over the brachial and crural arteries. The bruit de diable is often heard over the bulb of the jugular vein and the crural vein. All these phenomena are anæmic in their origin.

Buhl noticed that the blood had very little tendency to coagulation, and that none of the red blood-globules presented the nummular arrangement. Fabre found a diminution in the number of red blood-globules. Laschkewitsch noticed that, after a one-half-per-cent solution of sodium chloride was added to the blood, the red blood-globules presented amoeboid movements, processes of fission, and the development of prolongations. They also appeared paler than normal. Increase of the number of white blood-globules has been repeatedly described. E. Seitz reported the development of lymphatic leukæmia in a case of Addison's disease. Sohet and Van den Corput found pigment in the blood, but the patient had suffered from intermittent fever in the Tropics.

Serious nervous symptoms sometimes become prominent. Comparatively slight importance attaches to the pains which have been referred to, although they sometimes become so severe in the lumbar region that the patients can hardly walk except when bent over forward. The painful sensations in the muscles and joints appear to be primarily nervous in their nature. The situation becomes more serious when the tendency to syncope becomes marked. Certain patients have frequent attacks of convulsions. Paralysis of the limbs have also been noticed, likewise delirium and maniacal attacks. These conditions may be the direct cause of death.

In one of my cases, attacks of vaso-motor angina pectoris (vide Vol. I., page 144) developed; in another spontaneous gangrene and exfoliation of some of the toes.

The disease usually lasts several weeks, months, and even years (three years at the most). It generally advances uninterruptedly, and the patients finally die from increasing exhaustion or cerebral symptoms. Temporary improvement is observed occasionally, and it has even been claimed that the skin may grow lighter (?). No authentic cases of recovery are known. Acute exacerbations, attended with fever, sometimes occur, and lead to the erroneous assumption of acute Addison's disease. The affection may then assume a typhoid character, and the sensorium may be profoundly involved.

III. ANATOMICAL CHANGES.—The skin retains its dark color in the cadaver. Under the microscope, the cells of the rete Malpighii are found to be filled with pigment, which is partly diffuse, partly in the

shape of brownish or blackish granules. The pigment is sometimes found only in the cells which are immediately adjacent to the papillary body of the cutis; in other cases, it is present, though more scantily, in the higher layer of cells, but never in the horny cells of the epidermis. Free pigment is found here and there between the cells. The cutis proper may be free from pigment; in other cases it is found in its stellate cells.

The suprarenal capsules generally, though not constantly, present cheesy degeneration. They are enlarged, and sometimes weigh three hundred grains (normal weight five to eight grains). On section, we usually find a very considerable thickening of the connective-tissue capsule, and the latter sometimes has a transparent grayish appearance. In some cases, the parenchyma is entirely destroyed, and replaced by yellow, dry, little masses. In other cases, there are remains of approximately normal parenchyma, or the cheesy portions are surrounded by a reddish-gray tissue.

Virchow showed that we have to deal chiefly with cheesy, confluent tubercles. In cases which are not too far advanced, gray tubercles are seen around the cheesy nodules; it is possible, however, that the tubercles develop secondarily. The tubercles develop first in the cortical substance, into which the cheesy masses do not extend until a later period. But a few cases have been reported, in which caseation developed in the products of chronic inflammation in the suprarenal capsules.

Numerous giant cells are found in the recent eruptions of tubercle. The cheesy masses are composed of granular detritus, shrunken remains of cells, fat granules, granulo-fatty cells, and tablets of cholestearin. Calcification of the cheesy nodules has been repeatedly described.

As a rule, both organs are diseased. The tuberculosis of the suprarenal capsules may be the primary and sole affection, or similar changes may be found in the lungs, urogenital apparatus, intestinal tract, retroperitoneal lymphatic glands, or vertebrae.

Caseation of the suprarenal capsules is the most frequent cause of Addison's disease. In a much smaller number of cases it is the result of destruction of these organs by cancer, whether primary or secondary. The disease is still less frequently the sequel of waxy degeneration, chronic callous inflammation, hemorrhage or gummata of the suprarenal capsules. In a case described by Fraenkel, the disease seemed to be the result of embolism of the suprarenal artery.

In very rare cases the suprarenal capsules have been found entirely normal. Some authors believe, therefore, that the disease is the result of an affection of the sympathetic nerve fibres which pass to and from these organs, and attention has been accordingly directed to the solar plexus. The following changes have been noticed in this plexus: connective-tissue proliferation, fatty degeneration, pigment degeneration of the ganglion cells, atrophy of the ganglion cells and nerve fibres, nuclear proliferation in the sheaths of the fibres, etc. In other cases, however, the solar plexus and sympathetic have been found intact.

The explanation of this disease is attended by almost insurmountable obstacles. In our opinion the following is the most probable hypothesis. Addison's disease is the result of functional disturbances of sympathetic nerve tracts, whose most important clinical symptoms are changes in the color of the skin, disturbances of digestion, increasing weakness, and severe nervous symptoms. In the majority of cases these disturbances are the result of disease of the suprarenal capsules

(generally tubercular cheesy processes), which may exist independently or extend to the solar plexus. In rarer cases the plexus is affected independently of the suprarenal capsules. Finally, the disease sometimes appears to be the result of a functional affection of the sympathetic, which is not demonstrable anatomically. Disease of the suprarenal capsule is not always followed by Addison's disease; the latter does not develop unless the sympathetic is affected. The connection between the sympathetic and the formation of pigment in the skin is entirely obscure.

The following hypotheses have also been entertained: *a.* Addison's disease is not a morbid entity, since similar tegumentary changes are also found in other chronic diseases associated with cachexia. *b.* Holmgren believes that, as the result of suprarenal disease, more taurocholic acid is formed in these organs and passes into the blood; here it destroys red blood-globules and their coloring matter is deposited in the skin. *c.* Austin Flint and Gilliam claim to have found atrophy of the gastric glands, so that the disease is not connected with the suprarenal capsules. *e.* According to Jurgens, atrophy of the muscular coat of the intestines plays an important part in the etiology (?)

Experimental investigations have furnished purely negative results.

Among changes in other organs we may mention pigmentation of some of the viscera (this may occur under other circumstances); softening of the medulla of bones and formation of red marrow (a symptom of many cachexiæ); congestion of the white substance of the spinal cord, atrophy and pigment degeneration of the ganglion cells in the anterior horns (accidental appearances).

IV. DIAGNOSIS.—The diagnosis of the disease is easy. The tegumentary lesion is distinguished from cyanosis by the fact that it is not changed by pressure with the finger. The almost similar appearance produced by the prolonged administration of nitrate of silver (argyrosis) is recognized by the previous history. In intense jaundice the skin sometimes has a bronzed color, but the sclera then has a yellow color. A brown color, like that of a mulatto (nigrities) may develop in miserable tramps who have long suffered from clothes lice, but white cicatrices are visible, under such circumstances, in the pigmented skin.

If the diagnosis is positive, we may generally assume an affection of the suprarenal capsules as the cause of the disease.

V. PROGNOSIS AND TREATMENT.—The prognosis is unfavorable and the disease always terminates fatally.

The treatment must be confined to the use of tonics, stimulants, good diet, etc.

APPENDIX.

As we have heretofore mentioned, disease of the suprarenal capsules and Addison's disease are by no means convertible terms. Extensive lesions of these organs sometimes remain latent. Hemorrhages into the suprarenal capsules, especially in the new-born, have been observed occasionally, and may give rise to sudden death.

INDEX.

- Abscess of the intestines, 123
 - liver, 195
 - stomach, 65
 - kidneys, 290
 - oesophagus, 39
- Acetone in gastric cancer, 81
- Addison's disease, 351
- Adenoma of the bladder, 338
 - kidneys, 303
 - liver, 220
 - stomach, 83
- Albuminuria, 243
 - febrile, 245
 - nervous, 245
 - physiological, 245
 - toxic, 245
- Amaurosis, uræmic, 288
- Ammonia, acid urate of, in urinary sediment, 324
- Amœba coli, 142
- Amœboid movements of red blood-globules in hæmaturia, 248
- Amœboid movement of white blood-globules in cystitis, 330
- Amyloid degeneration of liver, 213
 - stomach, 95
 - kidneys, 297
 - suprarenal capsules, 354
 - reaction, 213
- Amyloidosis hepatis, 213
- Anæmia of kidneys, 263
- Anchylostomum duodenale, 158
- Aneurism of the cystic artery, 228
 - gastro-epiploic artery, 100
 - hepatic artery, 228
 - renal artery, 308
- Angina, acute catarrhal, 17
 - chronic, 22
 - parenchymatous, 20
 - phlegmonous, 20
 - tonsillar, 23
- Anguillula intestinalis, 158
- Aniline colors in amyloid reaction, 214
- Aphthæ, 7
- Arterio-capillary fibrosis, 282
- Ascaris lumbricoides, 152
 - mystax, 155
- Ascites, 237
 - fatty, 238
- Ascites, chylous, 239
- Aspermatism, 346
- Aspermatozia, 346
- Atrophy of liver, acute yellow, 207
 - chronic, 201
 - gastric mucous membrane, 95
 - kidneys, 281
- Axis of intestines, torsion of, 128
- Balantidium coli, 143
- Bile-ducts, diseases of, 171
 - ascaris lumbricoides, 190
 - calculi, 181
 - cancer, 191
 - catarrh, 178
 - distomum hepaticum, 190
 - echinococcus, 190
 - fibroma, 191
 - fistula, 187
 - myoma, 191
 - purulent inflammation, 181
 - parasites, 190
 - stenosis and occlusion, 171
- Biliary acids, reaction of, 174
 - fistula, 181, 187
- Bilirubin infarction of kidney, 324
- Bladder, diseases of, 326
 - cancer, 335
 - catarrh, 326
 - distomum hæmatobium, 339
 - echinococcus, 339
 - foreign bodies, 339
 - gall-stones in, 339
 - hyperæsthesia, 342
 - leptothrix, 338
 - neuroses, 340
 - paralysis, 343
 - parasites, 338
 - sarcina, 338
 - spasm, 342
 - tumors, 338
- Blood tests, 250
 - spectrum, 249
- Bothriocephalus latus, 145
 - cordatus, 151
- Bright's disease, 268
- Bronzed skin, 351
- Buccal mucous membrane, catarrhal inflammation, 1

- Cæcum, inflammation of, 123
 Calculi, hepatic, 181
 renal, 315
 Cancer of intestines, 137
 Capsulitis, 270
 Caput Medusæ, 205
 Cardialgia, 101
 Cavernoma of kidneys, 303
 Cercomonas intestinalis, 143
 Cholæmia, 176
 Cholestearin calculi, 184
 pigment calculi, 185
 Changes in position of liver, 224
 stomach, 98
 kidneys, 307
 Cheyne-Stokes respiration in uræmia, 259
 Chlorosis, tropical, 158
 Cholangitis, catarrhal, 178
 suppurative, 181
 Choked disk, in cirrhosis of kidneys, 288
 Cholecystitis, suppurative, 181
 Cholecystotomy, 190
 Cholelithiasis, 181
 Cholera infantum, 114
 morbus, 112
 Chyluria, 254
 Cirrhosis hepatis, 201
 arterio-sclerotic, 201
 hypertrophic, 202
 monocellular, 203
 renal, 281
 senile, 201
 Cirsomphalos, 205
 Colic, flatulent, 168
 hepatic, 186
 renal, 319
 Colitis, 107
 Coprostasis, 127
 Cowper's glands, 351
 Cystic kidneys, 304
 Cysticercus cellulosæ of the intestines, 148
 of the peritoneum, 242
 liver, 224
 kidneys, 305
 Cystin calculi, 318
 Cystinuria, 257
 Cystitis, 326
 acute, 328
 chronic, 332
 Cystoplegia, 343
 Cystospasmus, 342

 Diarrhœa, fatty, 120
 nervous, 170
 Dilatation of œsophagus, 33
 stomach, 83
 Distomum crassum, 159
 hæmatobium in hæmaturia, 326
 in the bladder, 339
 in the renal pelvis, 326
 hepaticum, 190
 heterophyes, 159
 lanceolatum, 190

 Diverticula of bladder, 328
 œsophagus, 33
 Dropsy of gall-bladder, 190
 Duodenal ulcers, 136
 catarrh, 109, 121
 Dyspepsia, nervous, 104
 Dysphagia, inflammatory, 38
 paralytic, 49
 spastic, 50
 Dystopia renum, 307
 Dysuria, spastic, 342

 Echinococcus of peritoneum, 242
 kidneys, 305
 liver, 220
 ova of, 221
 Echinorhynchus gigas, 159
 Embolism of retinal arteries in cirrhotic kidneys, 289
 of renal artery, 296
 of mesenteric artery, 170
 Empyema cystidis felleæ, 191
 Endarteritis obliterans, 282
 Enteralgia, 168
 Enteritis, acute catarrhal, 105
 chronic, 118
 phlegmonous, 123
 polypoid, 119
 purulent, 123
 Enterodynia, 168
 Enteroliths, 124
 Enterorrhagia, 159
 Enterostenosis, 127
 Enuresis, nocturnal, 340

 Fæcal odor of pus in urine, 331
 of exudation in peritonitis, 232
 Fatty degeneration of liver, 211
 kidney, 299
 diarrhœa, 120
 Fibrin concretions in the renal pelvis, 316
 Fibrinuria, 255
 Fibroma of biliary passages, 191
 liver, 220
 kidneys, 303
 Filaria sanguinis, 254
 Flatulent colic, 168
 Floating kidney, 306
 Follicular ulcers of intestines, 107
 œsophagus, 38
 stomach, 75
 Friction murmur in peritonitis, 195, 233

 Galacturia, 254
 Gall-bladder, diseases of, 171
 cancer, 191
 calculi, 181
 dropsy, 190
 empyema, 191
 extirpation, 190
 faradization, 181
 tumors, 191
 Gall-stones, 181

- Gas, accumulation of, in peritoneum, 235
 inflammable, in stomach, 85
 Gastralgia, 101
 Gastritis, acute, 56
 chronic, 59
 croupous, 64
 phlegmonous, 64
 purulent, 64
 submucous, 64
 toxic, 66
 Gastrodynia, 101
 Gastroectasia, 83
 Gastromalacia, 95.
 Gastrorhexis, 96
 Gastroxynsis, 105
 Glomerulo-nephritis, 270
 Glossophyton, 14
 Gmelin's test for bile-pigment, 173
- Hæmatemesis, 53
 Hæmatin spectrum, 249
 Hæmatinuria, 252
 Hæmatoidin crystals in urinary sediment, 272
 infarctions, 324
 Hæmaturia, 246
 Hæmin crystals, 251
 Hæmoglobin spectrum, 249
 Hæmoglobinuria, 252
 Heller's blood test, 249
 Helminthiasis, 141
 Hemeralopia, 175
 Hemorrhage of intestines, 159
 Hemorrhoids, 164
 Hepatic artery, aneurism of, 228
 Hepatitis, suppurative, 195
 chronic interstitial, 201
 acute, diffuse parenchymatous, 207
 Hemialbumose in urine, 245
 Horse-shoe kidney, 307
 Hydatid tremor, 223
 Hydrencephaloid, 115
 Hydronephrosis, 308
 Hydrothionuria, 255
 Hydrops cystidis felleæ, 174
- Icterus catarrhalis, 178
 gastro-duodenal, 178
 hematogenous, 199
 hepatic, 171
 mechanical, 171
 menstrual, 179
 paralytic, 179
 Ileitis, 109
 Ileus, 130
 Impotence, 345
 Incontinence of pylorus, 94
 Indican test, 80
 Indigo calculi, 318
 Infarctions of kidneys, 296, 323
 Intestines, diseases, 105
 cancer, 137
 erosions, 107
 hemorrhage, 159
 intussusception, 130
- Intestines, inflammation, acute catarrhal, 105
 chronic, 118
 phlegmonous, 123
 invagination, 130
 occlusion, 127
 parasites, 141
 polypi, 141, 119
 stenosis, 127
 torsion, 130
 tumors, 137
 ulcers, 107, 119, 121
 Ischæmia of kidneys, 263
 Ischuria spastica, 342
- Jejunitis, 109
- Kidneys, diseases of, 258
 abscess, 290
 amyloid, 297
 atrophy, 282
 Bright's disease, 268
 calculi, 315
 cancer, 300
 changes in shape, 307
 cirrhosis, 281
 cloudy swelling, 299
 congestion, 265
 cystic, 304
 embolism, 296
 fatty, 299
 floating, 306
 gouty, 283
 hemorrhagic infarction, 296
 ischæmia, 263
 parasites, 305
 supernumerary, 308
 tumors, 303
 waxy, 297
- Lallemand and Trousseau's corpuscles, 351
 Large white kidney, 277
 Lead kidney, 283
 Leptothrix buccalis, 13
 Leucin in urine, 210
 Leucoplacia oris, 9
 Lime calculi, 318
 infarctions, 323
 Lipuria, 255
 Littre's glands, diseases of, 351
 Liver, diseases of, 191
 abscess, 195
 acute yellow atrophy, 207
 amyloid, 213
 cancer, 216
 changes in position, 224
 changes in shape, 225
 cirrhosis, 201
 cysticercus cellulosæ, 224
 echinococcus, 220
 fatty, 211
 floating, 225
 hyperæmia, 191
 red atrophy, 193
 tumors, 220

- Lymphatic glands, disease of mesenteric and retroperitoneal, 230
- Magnesia phosphates in urinary sediment, 86
- Marechal's test for bile-pigment, 173
- Melæna neonatorum, 163
- Melanuria, 254
- Merycismus, 100
- Metamorphopsia, 287
- Methæmoglobin, 249
- Mictus cruentus, 246
- Movable kidney, 306
- Mulberry calculi, 317
- Mycosis pharyngis leptothricia, 25
- Mycosarcoma striocellulare of kidneys, 303
- Myxoma of bladder, 338
kidneys, 303
liver, 220
- Necrosis of kidneys, 296
- Nematodes in intestines, 152
- Nephritis, acute diffuse, 268
chronic interstitial, 281
parenchymatous, 276
suppurative, 290
- Nephrolithiasis, 315
- Neurasthenia gastrica, 104
- Nutmeg liver, 193
- Nyctalopia, 175
- Œsophageal sound, 29
stenosis, 26
- Œsophagismus, 50
- Œsophagitis, 37
- Œsophagomalacia, 48
- Œsophagomycosis oïdica, 49
- Œsophagoscopy, 31
- Œsophagus, diseases of, 26
auscultation, 30
cancer, 41
diverticula, 35
dilatation, 33
hemorrhage, 44
inflammation, catarrhal, 37
corrosive, 40
phlegmonous, 39
paralysis, 49
perforation, 45
rupture, 47
softening, 48
sprue, 49
stenosis, 26
ulceration, 41
- Oidium albicans in stomach, 97
buccal cavity, 10
œsophagus, 49
- Oxalate of lime calculi, 317
infarctions, 323
- Oxaluria, 256
- Oxyhæmoglobin spectrum, 249
- Oxyuris vermicularis, 155
- Pancreas, diseases of, 229
cancer, 229
- Pancreas, hemorrhage, 229
inflammation, 229
- Paranephritis, 293
- Paratyphlitis, 123
- Pelvis of kidneys, diseases of, 308
calculi, 315
dilatation, 308
distomum hæmatobium, 326
inflammation, 310
tumors, 324
- Peptonuria, 243
- Perforation-peritonitis, 235
- Perihepatitis, 194
- Perinephritis, 296
- Peritoneum, diseases of, 230
cancer, 242
inflammation, 230
œdema, 237
parasites, 242
- Peritonitis, acute diffuse, 233
acute local, 235
diffuse chronic, 236
circumscribed chronic, 235
- Perityphlitis, 123
- Pettenkofer's test, 174
- Pharyngitis, acute, 17
chronic, 22
superficial, 20
parenchymatous, 20
lacunar, 21
- Phlebectasia hemorrhoidalis, 164
- Phleboliths in hemorrhoids, 165
- Phosphatic calculi, 317
- Piles, 164
- Platodes in intestines, 143
- Pneumo-peritonitis, 235
- Poikilocytosis, 80
- Pollutions, 347
- Portal vein, diseases of, 226
inflammation, 227
stenosis and occlusion, 226
- Proctitis, 121
- Propeptone in urine, 245
- Prostatorrhœa, 350
- Protozoa in intestines, 142
- Ptyalism, 14
- Puerperal peritonitis, 234
- Pyelitis, 310
- Pyelonephritis, 312
- Pylephlebitis, 227
- Pylethrombosis, 226
- Pylorus, dilatation of, 94
stenosis, 83
- Pyonephrosis, 312
- Pyrosis, 58
- Pyrocatechinuria, 254
- Renal artery, aneurism of, 308
calculi, 315
colic, 319
pelvis, tumors of, 324
- Resection of the œsophagus, 32
pylorus, 94
stomach, 82
- Retinitis albuminurica, 288
apoplectica, 288

- Round worms in intestines, 152
 Rumination, 100
 Rupture of stomach, 96
 oesophagus, 47
- Saccharomyces albicans of the oesophagus, 49
 Saliva, retention of, 17
 increased secretion, 14
 diminished secretion, 17
 stasis of, in aphthæ, 17
- Sarcina oris, 14
 urinæ, 338
 ventriculi, 86
- Sarcoma of bladder, 338
 intestines, 141
 kidneys, 303
 liver, 220
 stomach, 83
- Semen crystals, 350
- Sexual apparatus, diseases of, 345
 aspermatisms, 346
 azoospermia, 346
 impotence, 345
 prostatorrhœa, 350
 spermatorrhœa, 347
 sterility, 346
- Sialodochitis fibrinosa, 17
- Softening of oesophagus, 48
 stomach, 95
- Spirochæte plicatilis, 13
- Sprue, 10
- Sterility, male, 346
- Stomacacæ, 4
- Stomach, diseases of, 51
 abscess, 65
 atrophy, 95
 cancer, 75
 calcification, 95
 changes in shape, 98
 position, 98
 degeneration, 95
 dilatation, 83
 hemorrhage, 51
 hemorrhagic erosion, 75
 inflammation, catarrhal, 56
 purulent, 64
 toxic, 66
 fistula, 69
 foreign bodies, 98
 hypersecretion, 101
 parasites, 97
 peristaltic restlessness, 101
- Stomach, rupture, 96
 softening, 95
 tumors, 75
 ulcers, 67
- Stomatitis, catarrhal, 1
 ulcerative, 4
 aphthous, 7
- Stomatomycosis oïdica, 10
- Strongylus gigas, 325
- Suprarenal capsules, diseases of, 351
- Tænia cucumerina, 151
 flavo-punctata, 151
 madagascariensis, 151
 nana, 151
 saginata, 146
 solium, 146
- Tape-worm, 143
- Teichman's blood-test, 250
- Tenesmus vesicæ, 320, 328
- Thread worm, 155
- Tight-laced fissure of liver, 225
- Tormina ventriculi nervosa, 101
- Trematodes in intestines, 159
- Trichiasis vesicæ, 339
- Trichomonas intestinalis, 143
- Tricocephalus dispar, 157
- Typhlitis, 123
- Tyrosin in urine, 210
- Umbilication of cancer, 216
- Uræmia, 258
 theory of, 262
- Ureters, diseases of, 308
- Urhidrosis, 261
- Uric-acid infarctions, 323
 calculi, 317
- Urine, incontinence of, 340
- Urocystitis, 326
- Urosepsis, 331
- Vesical tenesmus, 320, 328
- Volvulus, 130
- Vomiting, periodical, 105
- Waxy liver, 213
 kidney, 297
- Whip worm, 157
- Wind colic, 168
- Winkel's disease, 252
- Xanthin calculi, 318
- Xanthopsia, 175

